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# Monthly Message

## Medical Education for National Defense (MEND)

The origin of the Medical Education for National Defense (MEND) Program dates back to 1951 with the appointment of a committee by the Association of American Medical Colleges to consider methods whereby material from the armed services could be made available to the medical schools: research; toxicology; preventive medicine; pest control; epidemiology; field, aviation, and submarine medicine; and environmental factors and human engineering—all of special import to the military and in some instances unique to the services. The committee consulted with the Department of Defense and in 1952–1953, five medical schools were selected for a pilot project with $15,000 allotted to each. Many of the schools feared that this would entail extra hours added to their overburdened curricula and that there would be dictation of subjects; therefore the program languished and it was difficult to obtain supporting funds. In the early days the program was carried out largely through the efforts of Dr. Stanley Olson, Dean at Baylor, and Brigadier General (then Colonel) Sheldon S. Brownton, USAF, MC, of this office, as chairman of the federal committee.

In 1954 the program was reoriented. It was realized that the former ROTC program, which was current in the medical schools, required personnel from the military in those that accepted it; it reached comparatively few students; it added hours to the curricula of the schools; and was primarily an expensive procurement program. A MEND Program, however, which was acceptable to the schools, would reach all of the students, would not require allocation of military personnel at the schools, would not be essentially a procurement program, and would be much cheaper in cost. This has proved to be the case. Therefore the program was changed and medical schools were added upon application at the rate of not more than 10 a year, with a member of the faculty in each school accepting the responsibility of coordination.

Numerous trips are made throughout the year and symposia are held at such places as Walter Reed Army and National Naval Medical Centers; Federal Civil Defense operations are witnessed; lecturers are provided; and the groups from the various schools visit military installations to learn about problems peculiar to military medicine and civil defense. On return to their schools they include into their own courses what they wish of what they have seen and heard.

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# Foreword

The *United States Armed Forces Medical Journal* is a monthly publication of professional and administrative information for medical personnel of the Department of Defense. The Assistant Secretary of Defense (Health and Medical) and the Surgeons General of the United States Army, Navy, and Air Force invite members of the regular and reserve medical services, the professional consultants of the military departments, and other physicians and health scientists with an interest in Department of Defense activities to submit manuscripts for publication in this *Journal*.

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# Toxicity of Propellant Fuels and Oxidizers

DAVID B. DILL, PH. D.
KEITH H. JACOBSON, PH. D.

A SYSTEMATIC STUDY of the toxicology of propellant fuels and oxidizers as well as of other potentially toxic materials of defense interest was widely recognized in the late 1940's as a prerequisite to the adequate solution of problems of environmental medicine resulting from new defense technology. Information on the toxicity of these chemicals and on the mechanisms of their toxic action, as well as development of hygienic standards and procedures for detecting and treating toxic injury, was clearly required if some of the newer propellants, hydraulic fluids, and fire extinguishers were to be safely handled. In some cases, discovery of a high degree of toxicity of a specific chemical would be sufficient cause for dropping that material from the development cycle and searching for a less toxic substitute.

In searching for a laboratory of sufficient size within the Department of Defense, it became clear that the staff and facilities for research in biochemistry, pharmacodynamics, toxicology, pathology, and clinical research were already present in the Medical Division at the Army Chemical Center, Maryland. Two projects ensued, and have resulted in studies of a large number of compounds, such as aniline, furfuryl alcohol, hydrazine, 1,1-dimethylhydrazine (UDMH), methyl hydrazine, diborane, pentaborane, decaborane, red fuming nitric acid, chlorine trifluoride, and 90 percent hydrogen peroxide among the propellants; and such as trifluoromonobromomethane and difluorodibromomethane among the fire extinguishers. The results of these investigations have been published in many original scientific reports; in addition, reviews, technical bulletins, and safety and health manuals have been based in part on these scientific and technical reports.

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From the U.S. Army Chemical Warfare Laboratories, Army Chemical Center, Maryland. Presented on April 28, 1959, at the 30th annual meeting of the Aerospace Medical Association, Los Angeles, Calif.

The establishment of the project was a result of foresight on the part of several officers in the medical services. Major General Otis O. Benson, Jr., USAF, MC, contacted Brigadier General John R. Wood,* MC, USA, in August 1948 to inquire whether our laboratory could undertake research on the dangers of propellant fuels in present and prospective use. About the same time, General Wood received a letter from the Navy asking for similar investigations of hydraulic fluids. General Wood thought that a joint project would best answer the several needs, and so he arranged for a conference, which was held in Washington in October 1948. At that meeting, representatives of the Air Force, Navy, and Army informally agreed on a plan for our laboratory to investigate the health hazards of propellant fuels. To formalize the agreements, a project was drawn up and submitted to the Chemical Corps Technical Committee, and in March 1949 Project No. 4–16–17–01, "Health Hazards of Propellant Fuels and Casualty Treatment Therefor" was approved with a priority of 1B.

General Wood had been so certain of approval that he had the laboratory work well under way in late 1948. In this critical period of fiscal year 1949, one of his notable achievements was to obtain from the Chemical Corps $239,000 for the additions to our laboratories that were necessary to the program. This insured our capability, as far as facilities were concerned, to undertake the toxicologic, physiologic, pathologic, biochemical, and pharmacologic studies contemplated.

By May 1949 it was clear that there would be a continuing need for research not only on propellants, but also on a wide variety of other chemicals used or being considered for use by the military services. A new project, No. 4–61–14–002, "Health Hazards of Military Chemicals," was approved by the Chemical Corps Technical Committee on 18 July 1949. At that time it was estimated that 35 people and $240,000 a year would be required, apart from post support and other overhead expenses provided by the 2nd Army. The financial support and personnel spaces were not immediately forthcoming—funds obtained in fiscal year 1950 amounted to $158,000, and it was not until March 1950 that an allotment of 15 additional personnel spaces needed to complete the complement was granted.

Three major conferences have been held. The first, held at Army Chemical Center in April 1950 and reported in Medical Division Special Report No. 3 "Conference on Health Hazards of Military Chemicals Program," [1] was attended by many in the military services

*At that time Colonel Benson was Chief of the Research Division, Office of the Air Surgeon, and Colonel Wood was head of the Medical Division, Army Chemical Center.

and by prominent scientists outside the Department of Defense. Research accomplishments were reviewed and advice on relative priorities for specific studies was sought. The second conference was held in March 1954. Two reports, one unclassified [2] and the other classified,[3] summarizing the information and discussions thereon, were issued. The most recent conference was held in January 1958 and again was summarized in an unclassified [4] and a classified [5] report. These conferences brought together many representatives of the military services and their contractors, and helped to bring about a wider understanding of our aims and accomplishments.

In addition, over 100 detailed reports and scientific papers have been issued. These were listed and categorized in May 1957; [6] a more complete and up-to-date list has been prepared and is available on request. Several papers of general interest have been presented at scientific meetings, such as Krop's "The Toxicity and Health Hazards of Rocket Propellants," [7] presented before the joint meeting of the American Rocket Society and the Institute of Aeronautical Sciences in January 1954.

We are proud to have had a part in preparing the Liquid Propellant Safety Manual,[8] which appeared in October 1958. This was issued by the Office of the Assistant Secretary of Defense (Research and Engineering), Advisory Panel on Fuels and Lubricants. Stephen Krop was chairman of this committee before leaving the government service in 1957, and since then the junior author has been a committee member. Indicative of the state of flux in this field, additions to the manual are now being prepared. Another important publication to which we have contributed is a joint service bulletin, "Health Hazards from Propellant Fuels and Oxidizers;" [9] this is published mainly for the medical departments.

The wide recognition given our accomplishments under this project has been gratifying. To cite one example, Major Stumpe recently surveyed the field of propellant toxicity [10] and his report contains an excellent bibliography of 40 references, more than half of them original investigations or reviews from this project.

We shall not repeat details of findings already published, but rather shall summarize the highlights of the toxicity of several important propellants. Work on hydrazine was reviewed by Krop [11] in 1954. Later results, together with some data on 1,1-dimethylhydrazine (UDMH), were published by Jacobson and associates [12] in 1955, and a summary statement was included in Stumpe's article.[10] The effects of chronic poisoning by hydrazine and UDMH have been investigated, but insufficient study of the chronic toxicity of methyl hydrazine has been performed to permit much discussion of that

compound. The most striking effect of chronic poisoning by hydrazine is the fatty degeneration of the liver;[11, 13] in addition, focal interstitial nephritis, anorexia, weight loss, and terminal convulsions were seen in animals repeatedly exposed to hydrazine vapor.[13]

O'Leary [14] has cited evidence that methylation of hydrazine accentuates the central stimulatory effects of the hydrazine moiety, but reduces the metabolic effects in terms of liver and blood changes. These latter effects are probably more important toxicologically because of the difficulty in controlling them with drugs, whereas the central stimulatory effects can be at least partially controlled with barbiturates. Anemia results from chronic poisoning by UDMH.

The Committee on Threshold Limits, American Conference of Governmental Industrial Hygienists, has proposed threshold limit values of 1 part per million (ppm) for hydrazine and 0.5 ppm for UDMH.[15]

Propellant oxidizers include chlorine trifluoride, fluorine, red fuming nitric acid, nitrogen tetroxide or nitrogen dioxide, and ozone—all potent irritants. Liquid oxygen is hazardous because of contact injury. Nitrogen dioxide and the tetroxide exist together in equilibrium; results reported on the effects of $NO_2$ were actually obtained on a mixture of $NO_2$ and $N_2O_4$, but calculated as $NO_2$. These irritant oxidizers have low threshold limit values, reflecting their high toxicity. Values published by the American Conference of Governmental Industrial Hygienists [15] are shown in table 1.

The demand for more thrust for the propulsion of rockets has led to the use of highly toxic chemicals; a similar situation has prevailed in the search for new fuels for use in air-breathing engines. This search led to studies of fuels containing boron and hydrogen. Boron hydrides are highly toxic and many of them have a complex mode of toxic action; most studies of the toxicity and mechanism of action of these compounds have been conducted in our laboratory. Another important source of toxicologic data on these boron compounds is the

**Table 1.** ***Threshold limit values of some oxidant vapors***

| *Oxidant* | *Threshold limit (p.p.m.)* |
|---|---|
| Chlorine trifluoride | 0. 1 |
| Fluorine | 0. 1 |
| Ozone | 0. 1 |
| Nitric acid | 10 |
| Nitrogen dioxide | 5 |

University of Pittsburgh, which has performed investigations under contract with our laboratory and, through the Callery Chemical Company, with the U.S. Navy Bureau of Aeronautics.

One class of these borane fuels is represented by HEF–2 and another by HEF–3 and HiCal–3. They are made up of compounds containing boron and hydrogen, and offer more energy per unit weight than the more commonly known hydrocarbon fuels such as gasoline and JP fuels. However, while the hydrocarbon fuels offer only a slight toxicologic hazard, as evidenced by many years of safe handling guided more by respect for their fire hazards than for their toxic hazards, the boron-based fuels are highly toxic as well as flammable.

These fuels, like the better known boron hydrides pentaborane ($B_5H_9$) and decaborane ($B_{10}H_{14}$), have a complex action involving the central nervous system, cardiovascular functions, and metabolic pathways. Large doses affect the central nervous system as evidenced by tremors and convulsions. Small doses have a depressant action. In animals to whom these fuels have been administered, blood pressure first increases and later decreases to shock levels; this is usually followed by death. A pronounced hyperglycemia is frequently seen in experimental animals, but the toxicologic significance of this effect is not well understood at present.[16] In some experiments with isolated rabbit hearts perfused with HEF–3, there have been significant decreases both in rate and in amplitude of cardiac contraction. Attempts to reverse these effects have not been highly successful, although intracardiac injections of nicotinic acid, nicotinamide, and epinephrine have partially reversed them.[17] HEF–3 administered into the vein or the peritoneal cavity often causes pulmonary edema or hemorrhage in the cat, rabbit and rat.[18] Our attempts to reverse other effects of poisoning have failed, with the possible exception of convulsions, which are sometimes controlled, for example, by short-acting barbiturates.

The high degree of acute toxicity of these fuels is demonstrated in table 2.[19]

**Table 2.** ***LD50 of boron compounds in experimental animals***

| *Animal species* | *Route* | *Purified component of HEF–3 and HiCal–3* | *HEF–2* |
|---|---|---|---|
| Rabbit | Intravenous | 6 mg/kg | 7 mg/kg |
| Rat | Intragastric | 40 mg/kg | 240 mg/kg |
| Rabbit | Cutaneous | 80 mg/kg | 1000–3200 mg/kg |
| Guinea pig | Cutaneous | 160 mg/kg | >3200 mg/kg |
| Rat | 4-hour inhalation | 23 ppm | 12 ppm |
| Mouse | 4-hour inhalation | 6 ppm | 11 ppm |

Insufficient work on which to base good estimates of threshold limit values has been done on the boranes. Observations to date tend to support our previous speculation [19] that these standards are not likely to be set at values higher than 0.05 ppm, and may well be set lower.

The military chemicals project was initiated to provide information on acute and chronic toxicity, type of toxic action, and mechanism of toxic action of materials of interest to the Armed Forces; such information is necessary to the understanding of the nature of toxic injury and thus to prevention of injury, and also to the development of diagnostic and therapeutic procedures. Toxicity studies have been highly useful in guiding the development of engineering equipment and protective equipment. Existing threshold limit values, also known as maximum allowable concentrations, for most of these military chemicals are based largely or entirely on chronic toxicity studies carried out under this program. These chronic exposures simulate the normal working day, 5 days a week. The unusual requirements of the military services, especially on shipboard, require possible exposure to contaminants on a continuous basis, that is, for 24 hours a day, 7 days a week. Two toxicologic investigations under such continuous exposure conditions, using 4 species of animals, have been conducted so far.

It is appropriate to mention the role this project plays in bioastronautics and in submarine medicine. Trace concentrations of some substances, though innocuous for short or intermittent exposures, may become hazardous if exposures last for days rather than hours, as is the case in nuclear-powered submarines, and as may be true in space vehicles.

Repeated exposures to propellant fuels and oxidizers are hazardous to those engaged in their manufacture, storage, and transport. We must also consider the hazards of toxic end products of combustion, especially when these are nonvolatile; not only man but plant and animal life in the vicinity of launching areas may be endangered. For example, although boron oxide has a relatively low mammalian toxicity, it is toxic to plants, a fact to be considered when selecting areas where motors using boron fuels are to be operated. We are all aware of the toxic hazards of carbon monoxide, but toxicologic characterization of some of the other combustion products awaits chemical characterization of these end products, and this information is difficult to obtain.

The safe handling of propellant fuels and oxidizers requires alert and informed medical and safety officers. The aim of our program in military chemicals is to perform necessary investigations so that these officials will have the proper information to permit them to prevent

toxic injury, and where preventive measures fail, to guide diagnosis and treatment.

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# Modification of Standard Plastic Transfusion Pack to Permit Collection of Plasma

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SEVERAL YEARS AGO, the United States went out of the plasma-collecting business. All stocks of plasma were considered contaminated with the virus of hepatitis, and the attack rate after infusion of pooled plasma was 20 to 25 percent.[1] Several synthetics were produced as "plasma substitutes" but none were free of pharmacologic blemishes.[2] Polyvinylpyrrolidone (PVP) is a plastic which is stored—perhaps permanently—by the reticuloendothelial system; no one knows what damage it might cause eventually. Dextran, a polymerized carbohydrate, causes prolongation of bleeding time in a considerable proportion of normal recipients, a propensity which must be regarded as undesirable in a substance intended for the wounded. Plasma itself was the ideal plasma substitute except for the virus, and for this reason a continuous effort was maintained to eliminate the virus. It proved to be a "tough customer." Temperatures great enough to kill it would coagulate the plasma proteins unless the plasma was mixed with stabilizers and the heat was applied over long periods of time. Serum albumin proved to be virus free but too costly. Various chemicals and various kinds of radiant energy, alone and in combination, were tried. Some of them were successful in destroying the virus, but they were expensive or caused plasma proteins to become antigenic or to produce febrile or allergic reactions in recipients. It was a discouraging problem. However, a relatively simple and satisfactory means of disposing of the hepatitis virus has been found.[3, 4] When plasma is stored in the liquid state at room temperature, the virus dies. It must be stored at least 6 months before the virus is surely dead, but at the end of that time the solution

From Walter Reed Army Institute of Research, Washington, D.C.

of human protein is safe and also effective as a plasma volume expander. It seems fairly certain that large-scale stockpiling of plasma can, and probably will be resumed. This confronts us with another problem, an economic one: where will we obtain the plasma and how much will it cost?

During World War II, blood for plasma was diverted from the blood collected for transfusion. A proportion of the units of whole blood was sent to processing plants where plasma was separated from red cells, mixed in large pools, bottled, frozen and dried. The cost of collecting and shipping these units of blood was borne by the plasma program so that the price of a unit of plasma included both the processing and the cost of procuring the starting material. The plasma was not separated at the collecting center for fear of contamination, so that there was the additional expense of refrigeration en route to prevent lysis of the red cells. Then, as there was no use for the red cells at the processing plant, they were thrown away, a loss which represented 80 percent of the protein in every unit of blood. (In 500 ml of bank blood, there are 15 grams of plasma protein and 65 grams of hemoglobin.)

It has been pointed out that plasma can be taken from the red cells at the collecting centers, if care to prevent contamination is exercised, so that the red cells can be used for transfusion. Such a program has been followed in several hospitals, but it has usually required a great deal of "education" to induce the hospital staff members to administer the "packed red cells" in place of whole blood. In many cases it is not desirable to do so; this is especially true of surgical procedures where lost blood should be replaced with whole blood rather than packed cells.

The purpose of this communication is to demonstrate that with equipment now available it is possible to obtain starting material for the plasma program without sacrificing any units of whole blood and without converting any units to packed red cells.

## THE SATELLITE POUCH

Our proposal involves the removal of about 70 ml of plasma from every unit of whole blood collected. Blood is routinely collected into an anticoagulant solution (ACD), the volume of which is 70 ml. This solution dilutes the blood, and as a consequence the hemoglobin and hematocrit are depressed. For example, the hematocrit of normal venous blood is about 45 ml per 100 ml. The hematocrit of bank blood is 37 or 38 ml per 100 ml. By removing an amount of plasma equal

to the volume of anticoagulant solution, 70 ml, the bank blood is restored to its original hemoglobin concentration and hematocrit. Such a unit of blood is a practical equivalent of unmodified bank blood. The plasma proteins are in the same concentration as in blood diluted by anticoagulant solution, but the hematocrit and hemoglobin concentration have been restored to normal (table 1). The loss from each unit amounts to about 3.5 grams of protein.

**Table 1.** ***Hemoglobin in blood pack before and after removal of plasma****

| Donor Hb gm/100 ml= 100% | Hb as percent of donor Hb | | Donor Hb gm/100 ml= 100% | Hb as percent of donor Hb | |
|---|---|---|---|---|---|
| | Pack after bleeding—% | Pack after plasma loss—% | | Pack after bleeding—% | Pack after plasma loss—% |
| 15.3 | 90 | 107 | 13.3 | 89 | 103 |
| 12.3 | 93 | 108 | 15.3 | 93 | 101 |
| 14.3 | 93 | 99 | | | |

*Of 30 units tested in this fashion, none varied from the first 5 shown above.

The safe and rapid removal of this increment of plasma is accomplished by a modification of the standard plastic blood transfusion bag (fig. 1). A small "satellite pouch" is hermetically attached to the bag by a length of plastic tubing. The tubing is closed by means of a steel shot plugged into the end; neither the anticoagulant solution nor the blood can move into the pouch prematurely.

Blood is drawn from the donor in the usual fashion. The unit of blood may then be centrifuged or placed in the blood bank refrigerator for 24 hours until the red cells have partially settled.* Two loose throw knots are placed in the tube between the bag and satellite pack and the steel shot is squeezed out of the end of the tube so that it falls into the bag (figs. 2 and 3). The satellite pouch is placed on one side of a laboratory balance, which is then balanced, and 70 grams of weights are placed on the other side (fig. 3). The bag of blood is gently squeezed so that the separated plasma flows through the tubing into the pouch. When the balance tips the tube is clamped. The throw knots are pulled tight to make a hermetic seal and the tube is cut between them (fig. 4). Transfer of the plasma has been accom-

*When units of blood become outdated, the entire amount of plasma may be transferred to the satellite pouch. If this policy is followed, it would be necessary to postpone removal of the plasma until the blood is ready to be removed from the blood bank, either for transfusion or discard.

plished in a completely closed system without any chance of contamination.

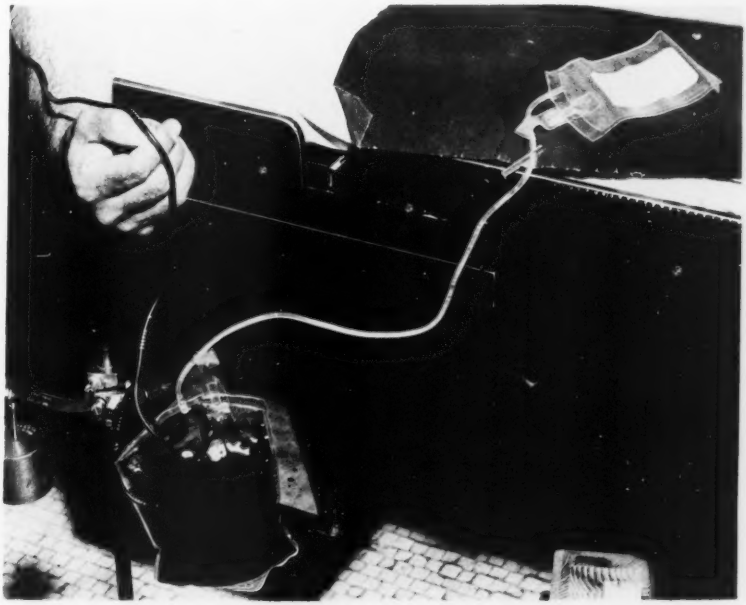

**Figure 1. A donor is being bled into a plastic blood transfusion bag. The satellite pouch, attached to the bag by plastic tubing, lies on the bench beside the donor.**

The plasma in the satellite pouch may be used in a number of ways. The pouch is equipped with an outlet port for attachment of a transfusion set, so that the plasma may be administered to patients who need plasma replacement rather than whole blood. It may be frozen immediately and stored, to be used when fresh plasma is indicated, as in the treatment of hemophilia. It may be shipped unrefrigerated to plasma processing plants to be used as starting material for the plasma program.

The satellite pouch has been tested on the transfusion service at Walter Reed Army Hospital. Five hundred units were filled, substituting the special bag with satellite pouch for the standard plastic bag. Seventy grams of plasma were removed from each unit the day after collection. The time required to collect the plasma averaged less than 5 minutes per unit. In a few instances the red cell sedimentation was

slow and the bags were centrifuged to clear the plasma. Measurement of the hemoglobin concentration was done on the first 30 bags. Three measurements were made (table 1): (1) the donor's venous blood,

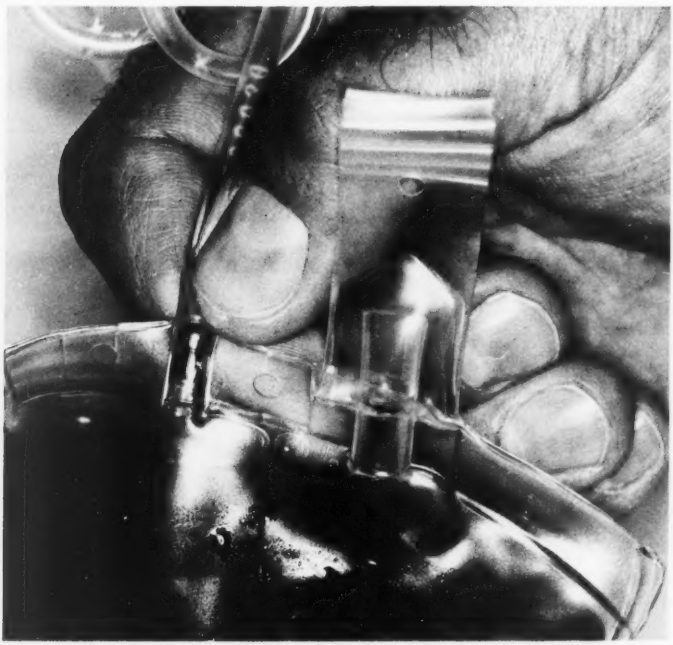

**Figure 2. A steel bead closes the tube leading to the satellite pouch. When ready to transfer plasma to the pouch, the bead is squeezed out of the tube and falls into the bag of blood.**

(2) the blood in the bag immediately after collection, and (3) the blood in the bag after the 70-gram increment of plasma had been transferred to the satellite pouch. The measurements confirmed the obvious—the blood was diluted by the 70 ml of ACD solution in the bag, so that the hemoglobin concentration was lower in the bag than it was in the donor's blood. Removing the increment of plasma restored the hemoglobin to "normal" in the bag.

The 500 units of blood were used by the medical and surgical services of Walter Reed Army Hospital without complaints and with only one comment. One of the officers in thoracic surgery remarked that patients who received large transfusions of this blood came into

the postoperative period with hematocrits a little higher than usual, around 48 ml per 100 ml instead of 42 ml per 100 ml.

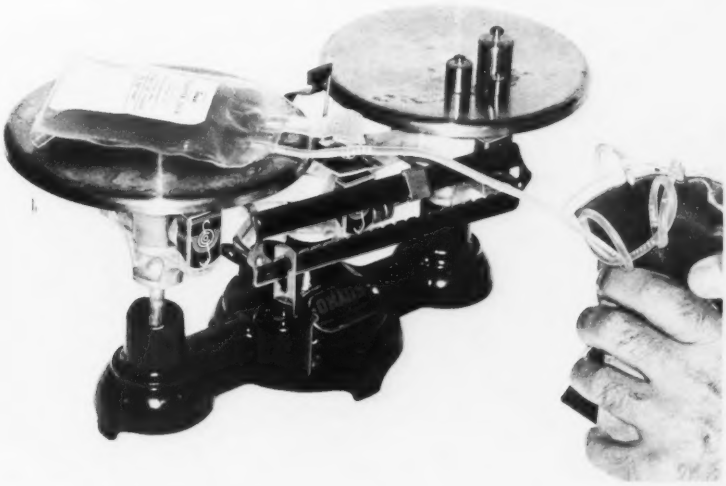

**Figure 3. The satellite pouch is placed on the pan on the laboratory balance, which is adjusted to counterbalance the weight of the pouch. On the opposite pan 70 grams of weights are placed, and plasma is squeezed from the bag into the pouch until the balance tips. Note that there are two loose throw knots in the connecting tube. When the transfer of plasma has been completed these knots are pulled tight to form a hermetic seal in the tube.**

The trial was a success. It demonstrated that the units of blood were not substantially altered by removal of a small amount of plasma. The collection of plasma was carried out without placing any great demand upon the blood bank personnel. The units of blood were accepted without complaint by physicians and surgeons alike. Most important from the point of view of those interested in the plasma program, 35,000 ml of plasma was obtained without sacrificing a single unit of blood.

## DISCUSSION

During the former plasma program the manufacture of 1 unit of dried plasma required 2 units of whole blood and cost about $27. About $15 of this amount represented the cost of collecting and ship-



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ping the 2 units of blood. Manufacture of 1 unit of serum albumin required 4 to 5 units of blood and cost about $45. Of this sum, about $35 represented the cost of the blood. It is obvious that the greater part of the cost of plasma or serum albumin represents the cost of collecting and shipping whole blood as the starting material. Plasma collected in this fashion costs about 4 cents per ml. Plasma collected in satellite pouches would probably cost about 1 cent per ml if extensive collections were instituted.

**Figure 4. The tube is cut between the knots. The transfer of plasma has been accomplished in the completely closed system without risk of contaminating either the plasma or the blood which remains in the bag.**

Usually it is difficult to obtain donations of blood in considerable numbers. It has been found that advertising and other kinds of social pressures are continually needed to counteract the public's apathy regarding requirements of the national blood program. Use of the satellite pouch will prevent a further complication of the problem. Otherwise, to provide for the plasma program, the national blood program would have to be expanded, or a competitive blood procurement program would have to be established. In either case the cost and difficulty of procuring blood would be increased. With the satellite pouch the blood program and the plasma program will be entirely compatible. There will be no requirement for expansion and little, if any, increase in cost.

## SUMMARY

The plastic transfusion pack is a standard item of medical supply for the Armed Forces and has gained wide acceptance in civilian transfusion services. It has proved to be superior to the glass bottle as a means for collection, storage, and administration of blood. A modification of the standard plastic pack has been developed which permits each unit of blood to be "taxed" 70 ml of plasma, an amount equivalent to the volume of the anticoagulant solution which is added to the blood during collection.

A small satellite pack is fused to the parent bag by means of a length of plastic tubing. The blood is drawn into the parent pack in the usual fashion and is placed in the refrigerator. When the cells and plasma become separated, the plastic tube is opened and the parent bag squeezed so that 70 ml of plasma run into the satellite. The tube is then clamped and cut. The loss of plasma restores the blood to its original hematocrit so that *it can be utilized as whole blood* rather than as packed red cells. This point is readily accepted by those who use the blood. By taxing each unit of blood a small amount, the procedure provides material for a plasma program without sacrificing any red cells or units of whole blood. The sterility of the blood and the plasma is not endangered because the transfer of plasma is accomplished in a completely "closed" system.

This method of collecting starting material for a natural plasma program has been given an extensive trial which demonstrated its practicality. If used on a broad scale it would materially reduce the cost of the plasma program.


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# Lupus Erythematosus Cells in Scleroderma

## Report of a Case and Analysis of the Phenomenon

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TRANSITIONAL SYMPTOMATOLOGY within the group of collagen vascular diseases may cause confusion and delay in precise diagnosis. The following report concerns a young woman with undoubted scleroderma who manifested positive L.E. (lupus erythematosus) cell tests on two occasions. Only a few years ago discussion of her case invariably centered on the specificity of the L.E. cell test. Today, particularly in view of recent experimental studies (vide infra), provocative questions arise regarding basic interrelationships in the group of collagen diseases.

### CASE REPORT

The patient (BNH 251027), a 21-year-old white woman, was admitted in August 1956 to this hospital for study and treatment of a disease thought to be typical of scleroderma. She had been transferred from the U.S. Naval Hospital at Key West, Florida.

Her illness had begun in January 1950, at which time she noted the presence of a discrete "spot," white and waxlike to the touch, just above the medial malleolus of the right ankle. It was little more than 1 cm in diameter when first noted, and several physicians who examined the lesion failed to determine its nature. She was eventually referred to a dermatologist who made a clinical diagnosis of scleroderma, and a therapeutic trial of Bistrimate (sodium bismuth triglycollamate) was begun. This medication was used in varying dosage over the next 2 years, but proved ineffective. The original lesion continued to increase in size, and by the middle of 1952, both legs showed rather symmetrically placed sclerodermatous plaques, which extended almost halfway to the knees. The dorsa of the feet were uninvolved.

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In June 1952, biopsy specimens were sent to two university centers for independent histopathologic interpretation. Pathologists at both centers reported that the tissue picture was compatible with the clinical diagnosis of scleroderma.

*Pathologist A.* "We noted subdermal fibrosis and hyalinization, with partial destruction of skin appendages. An inflammatory component was noted, and also foci of calcification. It was our feeling that the picture was compatible with a diagnosis of scleroderma."

*Pathologist B.* "The overlying epidermis is pigmented and somewhat atrophic. The dermis is thickened and composed of dense collagenous tissue. This surrounds and compresses scant dermal adnexae. Small sweat glands are incorporated in the sclerotic dermis. In addition, there is a chronic inflammatory reaction, predominantly perivascular and lymphocytic. The walls of the vessels are thickened. It is felt that the histologic appearance of this skin segment is consistent with a diagnosis of scleroderma. It is not possible on histologic grounds alone to distinguish the clinical 'circumscribed' and 'generalized' varieties."

Following establishment of a tissue diagnosis, the patient traveled widely throughout the country, seeking the aid of many physicians, but rarely staying with any one for an appreciable length of time. There were several short periods of hospitalization, and she was repeatedly presented to medical students as a "classic case of scleroderma." In October 1952, cortisone therapy was begun, and continued on an interrupted dosage schedule for 8 months, without noticeable improvement. Artane (trihexyphenidyl) hydrochloride was added to the cortisone regimen for an additional 4 months, again without apparent effect. The disease progressed inexorably; leg involvement extended to the knees, and the arms became involved from the wrists to the elbows. Although there was very marked stiffness of the joints, Raynaud's phenomenon was not noted. Subsequently, the disease spread over the upper arms and legs, and the hands became involved. Patches of morphea appeared over the pelvic girdle and trunk. In July 1954, the dorsum of the left foot was scratched, and an indolent ulcer developed which required 3 years to heal. During the worst period, extensor tendons were exposed over the entire dorsum of the foot. The use of crutches and consequent flexion of the left knee resulted in ankylosis of that joint.

During the period from May to December 1955, the patient was treated with relaxin. She noted no improvement. However, the skin lesions, which had been "dead white" in color, became definitely bronzed at this time.

In January 1956, prednisone therapy was begun, and in addition the patient received six injections of ethylenediaminetetraacetic acid. Her condition seemed unchanged.

At the time of admission to this hospital, a pertinent feature of the physical examination included atrophy of the skin of all extremities, the process being more marked distal to the elbows and knees, but present in lesser degree proximally. The skin was distinctly brown in color, firmly adherent to the tissues below, and hard and waxy to the touch. There were flexion contractures of the fingers of both hands and of the left knee. Movement of the left ankle was restricted, and the patient was ambulatory only with crutches. Large areas of depigmented and atrophic epithelium were present over the back, and there were distinct sclerodermatous plaques over the pelvic girdle. There were many trophic ulcers over the dorsum of the left foot and ankle. The patient was 3 months pregnant. (Figs. 1–4.)

Extensive laboratory examinations were performed during the period of hospitalization. Representative studies included a white blood cell count of 6,850 per cu mm, with 69% neutrophils, 3% band forms, 19% lymphocytes, 2% monocytes, and 7% eosinophils. Hemoglobin was 11.5 grams per 100 ml of blood;

**Figure 1. Muscular atrophy and pigmentation of right hand and forearm, and flexion contractures of fingers. Healed ulceration on radial aspect is secondary to the application of a commercial cosmetic foundation with which the patient had attempted to conceal a discrete sclerodermatous plaque.**

**Figure 2. Extreme muscular atrophy of the lower extremities, flexion contracture of the left knee secondary to the use of a crutch, and trophic ulceration of the dorsum of the left foot.**

hematocrit 36 ml per 100 ml. The sedimentation rate was 12 mm per hour, by the Cutler method. A catheterized urine specimen was straw colored, and neutral in reaction. Specific gravity was 1.016. Neither sugar nor albumin was present in the urine. A few epithelial cells were seen on microscopic examina-

tion. The serologic test for syphilis was negative. A direct Coombs' test was negative. An electrophoretic pattern of serum proteins, obtained while the patient was on steroid therapy, was within normal limits. Total protein was

**Figure 3. Areas of depigmented and atrophic epidermis of back, and discrete plaques of scleroderma over the pelvic girdle.**

5.8 grams per 100 ml of blood, with 4.0 grams of albumin, and 1.8 grams of globulin. The direct van den Bergh test showed 0.04 mg per 100 ml of bilirubin, the indirect 0.14 mg per 100 ml. Cephalin flocculation was negative at 48 hours. Thymol turbidity was 1 unit; alkaline phosphatase 1.4 Bodansky units. Prothrombin time and control were both 13 seconds. Total cholesterol was 160 mg per 100 ml with 77% esters. Blood urea nitrogen was 12 mg per 100 ml; fasting blood sugar 70 mg per 100 ml; calcium 9.7 mg per 100 ml; phosphorus 3.8 mg per 100 ml. The differential titer of the hemagglutination test for rheumatoid

**Figure 4. Trophic changes and ulceration of the dorsum of the left foot.**

**Figure 5. An L.E. cell from 1 of the 2 positive preparations. (x 1000)**

arthritis was 1:4. Lupus erythematosus cell preparations, prepared by the method of Snapper and Nathan,[1] were positive on two occasions (fig. 5).

Roentgenograms of the chest and esophagus revealed no abnormalities. An upper gastrointestinal series was normal, as was the follow-up study of the small bowel. Intravenous pyelograms showed a normal upper urinary tract. Roentgenograms of the ankle and left knee showed demineralization of all visualized bones, and diminution of the soft tissue shadows overlying the osseous structures. There was narrowing of the interphalangeal joint spaces in both hands.

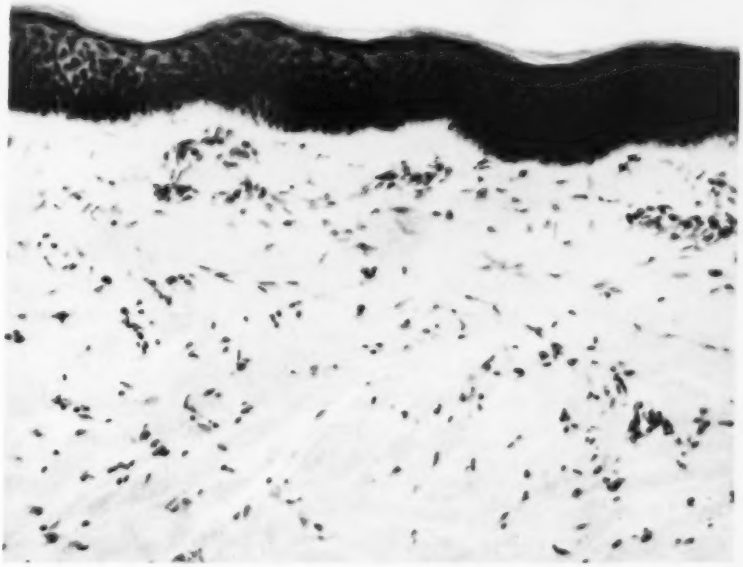

**Figure 6. Skin biopsy, showing loss of rete pegs, atrophy of dermal appendages, and heavy collagenization of the dermis. (x 150)**

The electrocardiogram was normal. Studies of pulmonary function indicated that the maximal breathing capacity was in the low normal range.

A repeat biopsy was not performed because the last site of excision had required 10 months for healing, making the patient reluctant to grant permission. However, both the previous biopsy specimens were reviewed by pathologists at this hospital, and they concurred in the opinion that the histopathologic picture was compatible with the clinical diagnosis of scleroderma (fig. 6). The L.E. cell preparations have been presented without history to several disinterested pathologists and hematologists, and have been considered positive by all who have seen them.

## COMMENT

Scleroderma has become such a well-recognized clinical entity that individual case reports hardly warrant publication unless they present some features of unusual interest. The case presented is of particular interest in several respects. The observation of a positive L.E. phenomenon in this disease is rare; the authors know of only two detailed reports of similar instances [2, 3] and brief references to two others.[4, 5] It has been possible to document 62 individual instances in which the L.E. phenomenon has been absent in patients with scleroderma,[6–12] and indeed there may be more. Corroborative statements of some authors were excluded when it was not clear whether they were writing from personal experience.

The rarity of the reported observation, however, is of minor importance, and now, having been mentioned, can be dismissed without further elaboration. Of much more compelling interest are the questions that arise regarding the specificity of the L.E. phenomenon, and the interrelationship of the several diseases in the so-called collagen vascular group.

Ever since the discovery by Hargraves, Richmond and Morton [13] of the L.E. cell in the marrow of patients suffering from systemic lupus erythematosus, the demonstration of such cells has been considered diagnostically specific. There have been several large-scale surveys attesting to the reliability of the L.E. test, and many physicians attribute a degree of specificity to the phenomenon that they would deny to other clinical laboratory procedures. Thus, in a recent survey [14] among 20 hematologists, all of whom had a special interest in the L.E. phenomenon, 15 found the test to be 90 to 95 percent reliable. Conley found the L.E. test to be more reliable than the Wasserman test, and felt that the reported lack of specificity was due to failure to "adhere to the rigid criteria in identifying L.E. cells." [15] Similarly, Dubois [16] has stated " . . . false positive tests do not occur, with the possible exception of the hydralazine syndrome, which may be the chemical induction of systemic lupus erythematosus." Harvey and associates [6] were unable to demonstrate the L.E. phenomenon in 663 instances when testing persons with diseases other than systemic lupus erythematosus. The consensus would appear to be summed up in the statement of Weiss and Swift,[7] " . . . the presence of clumping, rosettes, and the typical L.E. cell may, in the present state of our knowledge, be taken as conclusive evidence of systemic lupus erythematosus."

Nevertheless, the L.E. phenomenon is sometimes positive when the diagnosis of lupus erythematosus is clinically doubtful,[9, 17] and there

is a growing list of disease entities other than systemic lupus erythematosus in which typical L.E. cells have been reported. This list includes multiple myeloma,[7, 18] pernicious anemia,[19] dermatitis herpetiformis,[19] leukemia,[7] moniliasis,[20] Senear-Usher syndrome,[21] glomerulonephritis,[22] during cortisone withdrawal,[23] penicillin hypersensitivity,[24, 25] hydralazine toxicity,[26-29] miliary tuberculosis,[30] chronic viral hepatitis,[31, 32] post necrotic cirrhosis,[33] discoid lupus erythematosus,[7] serum sickness following tetanus antitoxin,[34] periarteritis nodosa,[4, 35] dermatomyositis,[4] thrombohemolytic thrombocytopenic purpura,[36] classic rheumatoid arthritis,[37] and during butazolidin therapy.[38] Haserick [39] has indicated that the L.E. phenomenon can be simulated by materials of fungal origin. Inderbitzen [40] has produced structures resembling L.E. cells with polyvinyl alcohol-polysulfonic acid ester, and normal gamma globulin. In essence, objections to all of these reports fall into three categories: (1) errors in original diagnosis, (2) nonrecognition of coexistent systemic lupus erythematosus, and (3) failure to adhere to rigid criteria in the identification of the L.E. cell.

It is possible, of course, to offer alternative viewpoints to the objections raised. There can be no doubt that in some of the reported cases the diagnosis of systemic lupus erythematosus was missed, and the observation of a positive L.E. phenomenon was, in fact, a reflection of the unrecognized disease. Indeed, systemic lupus erythematosus may be so vague in its clinical expression that a prolonged period of observation is necessary before diagnostic features are definitely manifest. It would appear unjustified, however, to dismiss the clinical observations of men with prolonged experience, contrary laboratory data notwithstanding, and assume that all previous reports of positive L.E. phenomena in diseases other than systemic lupus erythematosus were questionable. Several of the case reports, particularly those in which necropsy was performed, are convincingly documented. The suggestion of failure to adhere to rigid criteria in the identification of L.E. cells is not tenable today, since knowledge of pseudo L.E. phenomena, nucleophagocytosis, tart cell formation, and leukoagglutination is commonplace.

Interrelationships within the collagen vascular group of diseases have long been speculative. Kampmeier [41] has suggested that they represent expressions of a fundamental tissue process. Beigelman, Goldner and Bayles [42] place systemic lupus erythematosus and scleroderma in the same pathogenetic group, the former deriving from a severe, rapid connective tissue reaction, with edema and necrosis predominating, and the latter representing a slow, generalized mesenchymal response, with fibrosis and sclerosis predominant.

There is increasingly more persuasive evidence indicating that certain disease states may be manifestations of autoimmunization, although precise mechanisms are as yet obscure. Within this group might be mentioned certain of the hemolytic anemias, idiopathic thrombocytopenic purpura, Schönlein-Henoch purpura, systemic lupus erythematosus, thyroiditis, Sedormid and quinidine thrombocytopenia, the hydralazine syndrome, and perhaps those instances of penicillin hypersensitivity associated with a positive L.E. phenomenon.

Strong support for the view that the L.E. phenomenon may represent autoimmunization against leukocytes has recently been advanced. Mellors, Ortega and Holman [43] have observed that the nuclei of leukocytes undergoing transformation to form lupus erythematosus cells develop fluorescence when stained with a specific antibody for human gamma globulin, and that such fluorescence can be inhibited by prior absorption of the fluorescent antibody stain with human gamma globulin. Similarly, Bardawil and co-workers [11] have observed a specific reaction between cellular nuclei and the gamma globulin fraction of the sera of patients with systemic lupus erythematosus, scleroderma, dermatomyositis, and certain cases of rheumatoid arthritis presenting positive L.E. cell reactions. They feel that the affinity between serum gamma globulin and desoxyribonucleic acid (DNA) protein may reflect a natural phenomenon basic to these disease entities, and they voice the reasonable suspicion that all may be manifestations of a common disease process, initiated by sensitization with either intrinsic or extrinsic nucleoprotein.

In the case that we have presented, we find ourselves unable to accept a clinical diagnosis of systemic lupus erythematosus. We have had the opportunity of following the patient through several periods of hospitalization, and on an outpatient basis ever since her initial admission. We have seen her develop striking facial scleroderma. Although skin changes resembling scleroderma can be found in patients with systemic lupus erythematosus, they are not nearly so pronounced as those presented by this patient. The course of her illness, since its beginning 9 years ago, the development of the typical dermal lesions of scleroderma and their histopathologic confirmation cast doubt on any diagnosis other than scleroderma. The L.E. cell preparations, positive on two occasions, may indeed reflect a basic and fundamental relationship between systemic lupus erythematosus and scleroderma, but they do not in themselves serve to establish a clinical diagnosis.

## SUMMARY

The case report of a young woman with scleroderma, who manifested a positive L.E. cell phenomenon on two occasions, is presented. It is suggested that the observation of L.E. cells may reflect a fundamental relationship between scleroderma and systemic lupus erythematosus, but that such cells do not serve for the establishment of a diagnosis that is clinically doubtful.

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## THE PHYSICIAN AND THE MORTICIAN

Physicians do not like the odor of the tombstone around their work and many doctors shy away from the persistent social and professional contacts with funeral directors. This, however, is unrealistic reasoning. Funeral directors, as individuals, are no doubt jolly fellows who make very good social companions. More important than that, however, the funeral director can be a friend in need to the physician not only at the time of a death among his patients but also as an ally in some of the doctor's legal, administrative and legislative battles. . . . In the antiquity of their calling, the morticians need not take a back seat to anybody. Embalmers had achieved professional status in Europe and Asia at a time when many branches of medicine were still at the stage of a skilled craft. Morticians have been the victims of an understandable prejudice against them because no one likes to be reminded of death. For this reason, they had been denied the professional recognition which has been awarded to dieticians, nurses, pharmacists and psychologists. Yet, when the chips are down, it must be conceded that the professional mortician is indeed a practitioner of a very ancient and skilled science. Mark Twain used to say that the sign of a good life was when even the undertaker was sorry that the man died. As usual, Mark Twain was a wise man.—Funeral Director: Friend in Need (editorial). *The Journal of the Medical Society of New Jersey*, August 1959.

# Relation Between Certain Preservice Factors and Psychoneurosis During Military Duty

MERRILL ROFF, PH. D.

THIS IS THE THIRD REPORT of a project on the prediction of adjustment in relation to military service from preservice information recorded during childhood and adolescence in guidance clinic case histories. The basic aim of this work is to determine what types of preservice maladjustments and personality problems and associated background factors, as described when they were occurring rather than retrospectively, are significantly related to, and predictive of, maladjustments of various kinds in relation to military service.

All the male individuals from the two main guidance clinics in Minneapolis and St. Paul have been followed in terms of their military service records in the Selective Service system, in national records depots, and at the Veterans' Administration. An introductory report, describing these and other samples in some detail and indicating the types of criterion data available and the frequencies falling in various criterion categories, appeared as the first report in this series.[1] This also included a preliminary prediction study, global both as to method of prediction and as to outcome (satisfactory, unsatisfactory), which indicated that there is a substantial relationship between the preservice case history information and subsequent adult adjustment.

Once it had been found that global predictions of gross outcome could be made at a level high enough to be interesting, further work was directed toward the more analytic prediction of more specifically defined outcomes, beginning with the prediction of in-service psychoneurotic reactions.[2] In order to avoid the risk of losing the pre-

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dictability found with the global approach, subsequent procedures were aimed at moving gradually downward from global appraisals, rather than going at once to a completely analytic approach. It was found that youngsters who antagonized and were disliked by their associates to an unusual degree (as reported by persons, particularly in the school systems, who had had an opportunity to observe their behavior in relation to their peer group) were those who had psychoneurotic difficulties while in military service some years later. The present report describes a successful replication of this work on a new sample. It also presents additional information concerning the relation between psychiatric comments during preservice clinic treatment and subsequent outcome in service.

## METHODS AND MATERIAL

The work described in this report is a direct replication, on a second sample, of the predictive procedures described in the preceding report.[2] The sample described in that report contained 110 persons, all of whom had been dealt with the child guidance clinic of the Minneapolis schools during their grade or high school years, and who were in service during World War II. Half of the 110 subjects, the psychoneurotic group, had been medically diagnosed as having difficulty of a psychoneurotic kind (anxiety state or reaction, neurasthenia, conversion hysteria or conversion reaction, obsessive compulsive reaction, reactive depression, and mixed or unspecified) leading either to a medical discharge or, in a small minority of cases, to hospitalization followed by a return to duty. The other half, which served as a control group, was a "good" group selected as having reached and kept a grade of sergeant or higher, without history of disciplinary or mental health trouble at any point following entrance into service. The two groups were matched in preservice intelligence level, from information contained in their original case histories.

The sample used in the present study contains 98 cases, half psychoneurotic in service and half control as defined above, consisting of St. Paul cases and of Minneapolis cases more recent than those used in the preceding report. The psychoneurotic and control groups were selected in the same manner as before, and were similarly matched in preservice intelligence level.

## PROCEDURE

The procedure developed earlier [2] consists, briefly, of abstracting all items of information in the case histories which indicate the nature



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of peer-group adjustment and relationships in the period covered by the history, and of applying a set of rules for evaluating these. These rules for readers (who were first-year graduate students) evaluating the abstracted items related to three headings, were:

*Chronology.* The date was carefully retained for all abstracted information. In case of contradiction between earlier and later information, other things being equal, greater weight was to be given to the later information.

*Informants.* It was found desirable to accompany all abstracted items by the category of the person giving the information. A priority list of informants was developed, with instructions to the readers to examine first the data from informants with highest priority and to keep working down this list as long as, and only as long as, it was necessary to reach a decision.

**Priority List of Informants**

1. Persons outside the family who had an extended opportunity to observe him in a peer-group situation—that is, personnel of school, camp, scout organizations, et cetera, when quoted directly.
2. Visiting teacher or case worker when summarizing information from persons in category 1, without specific quotation; clear and definite formal diagnostic statements by psychiatrist which relate to social adjustment.
3. Family members, except for favorable statements by mother, which were not abstracted.
4. Statements about social adjustment by patient in interview, and statements by psychiatrist, except as noted above; comments about personality by psychologist based on impressions obtained during a mental testing session.

*Content.* A guide list for information to be evaluated as positive, neutral, or negative in appraising peer-group adjustment was developed. Positive items included such things as "all signs of liking by the general peer group" and "not a behavior problem in class or on the playground." Neutral items included such things as "shortage of friends without specific indications of being disliked; indifferent to other youngsters or they are indifferent to him" and "disciplinary problem in or around school without adverse reaction from other youngsters." Negative items included such things as "all signs of active dislike by the general peer group; do not count single fights or feud with a specific youngster, but count enough quarrels to get the boy labeled as quarrelsome" and "disciplinary problem in or around school with consequent adverse reaction from other youngsters."

Working with these three sets of guide rules concerning chronology, informants, and content, readers appraised the peer-group status indicated by the abstracts from the histories. Judgments of "good" or

"poor" were to be made only if the reader felt reasonably certain of a judgment. If the information seemed incomplete, unclear, or inconsistent, a response of undecided was to be made. When the results of these appraisals, which were completely "blind" with respect to outcome in service, were compared with status in relation to service, table 1 results.[2]

**Table 1.** ***Evaluations of preservice status in relation to outcome in service: earlier sample***

| Outcome | Appraisal | | |
|---|---|---|---|
| | Good | Undecided or neutral | Poor |
| Good | 27 | 21 | 7 |
| Psychoneurotic | 6 | 21 | 28 |

$x^2 = 25.97$
$P = < .001$

When the same procedure was applied to the second sample of 98 cases, the results shown in table 2 were obtained.

**Table 2.** ***Evaluations of preservice status in relation to outcome in service: new sample***

| Outcome | Appraisal | | |
|---|---|---|---|
| | Good | Undecided or neutral | Poor |
| Good | 27 | 11 | 11 |
| Psychoneurotic | 7 | 8 | 34 |

$x^2 = 23.99$
$P = < .001$

It may be seen by comparing the two tables that the ratio of successful predictions for the "good" and "poor" appraisals are similar. In each case the number of cases predicted "poor" who were later psychoneurotic is at least three times the number with "good" outcome. Cross-validation can be considered satisfactory.

## PREDICTIVE SIGNIFICANCE OF EARLIER PSYCHIATRIC EVALUATIONS

The preceding report also presented a comparison of psychiatric comments in the case histories with outcome in service. These comments were based not only on all that was known about the boy by the clinic at the time of first interview, but also, in many cases, on a substantial number of psychiatric interviews extending over a period of years. In many of the cases considered seriously deviant by the psychiatrist, some departure from normality was also clearly apparent to the peer group and teachers.

Analysis of these comments, which are present in each history, indicates that there are three kinds of comments which are ineffective in differentiating between cases in terms of later adjustment. One type of these may be called "contingent" predictions; in them, a hope is expressed that if some presently adverse factors in the situation change somewhat, an improvement might be expected. For example: "If he could be separated from his present bad home situation, he might get along all right."

A second kind of unpredictive comment is a statement of what the boy was considered to be adjusting to—sometimes in terms of a "psychodynamic" interpretation, sometimes in a recapitulation of factors in the home or elsewhere which seemed contributory to the problem. For example: "He evidently has a feeling of inferiority brought about largely by his mother's overindulgence and failure to encourage the development of self-confidence and independence. His problems have probably developed in part as defense reactions." "His temper outbursts are reactions to frustrations which probably have been developed in the home."

A third type of comment, unpredictive in relation to later psychoneurosis, is the "relative" comment. One form this may take is a statement that the individual is "improved," without indicating that he is in good condition. Other statements that may be similarly classified are that "he is making as good an adjustment as could be expected"; "he does not give such an impression of disturbance as he did in the previous interview"; "there are no really outstanding conflicts."

When statements of these three types are disregarded, there remain the definitely diagnostic judgments by the psychiatrist which appear about as effectively predictive in the present sample as they did in the earlier one. These are found in only a minority of cases, but when they occur, they are worthy of notice. Table 3 gives a case-by-case summary of the 22 of the present 98 cases in which definite comments, positive or negative, were made.

**Table 3.** ***Early psychiatric comments and subsequent service experience***

| Case No. | Psychiatric comment | Experience in service |
|---|---|---|
| | *Unfavorable comment with unfavorable outcome* | |
| 1 | He leaves the impression of being prepsychotic. The depression of a few months ago followed by his present hypomanic behavior suggests the possibility of a manic-depressive psychosis. As yet there has been no evidence of hallucinations or delusions. He is sexually conflicted because he has been sought after by boys for sexual purposes. There are two problems to be dealt with—that of mental illness and that of delinquency. | In service 14 months; cook, one reduction (Pfc. to Pvt.); medical discharge, psychoneurosis, severe, manifested by obsessive-compulsive reaction and episodes of acute anxiety with depression, emotional instability, latent homosexual trends, immaturity and pathologic lying. |
| 2 | Age 13: He is an unstable and very likely neurotic boy with anxieties and phobias which he may discuss in detail later on.<br>Age 15: His plans are so untenable that he leaves the impression of having little close touch with reality. A diagnosis of prepsychosis cannot be made with sureness. He does not appear to have become any more advanced in psychotic behavior since he was first seen.<br>Age 16: He still leaves the impression of being prepsychotic. In view of the fact that he has been this way for so long it is more likely that he is just an unusually bizarre person with abnormal character traits. | In service 9 months; Pvt., basic soldier. Medical discharge, psychoneurosis, mixed; has grandiose ideas concerning his abilities, and maintains a pseudointellectual attitude; showed marked effeminacy in mannerisms, speech and behavior. |
| 3 | It is quite obvious that he has not improved a great deal, and is the same concerned boy. He continues to leave the impression of being neurotic. | In service 14 months; Cadet midshipman. Medical discharge, psychoneurosis, neurasthenia. |
| 4 | He gives the impression of being a neurotic boy. He felt very sorry for himself and left the impression of being quite unstable. | In service 42 months, no overseas service. Cpl., airplane and engine mechanic. Hospitalized 6 weeks for neurasthenia and returned to duty. Later hospitalized for 3 months, psychoneurosis, anxiety, and returned to duty. |
| 5 | He is an unusually tense, restless, talkative, exhibitionistic adolescent filled with anxieties. He left the impression of being quite distinctly neurotic. | In service 31 months, no overseas service. T/4, surgical technician. Medical discharge for psychoneurosis, severe, manifested by severe anxiety and nervousness. |
| 6 | He is a neurotic delinquent. He left the impression of being quite panicky. Perhaps it was because of his marked emotional disturbance that he appeared to be possibly prepsychotic. | In service 3 months. Became unfit for duty after only 6 days of service. Medical discharge following hospitalization for psychoneurosis, mixed, psychasthenia and anxiety, constant preoccupation with fear of death and safety of relatives, irritability and insomnia; a preclinical dementia praecox. |
| 7 | Age 10: By degrees he is admitting a pleasure he gets through exhibitionism and being used passively as a sex object by other boys.<br>Age 12: It is quite obvious that he is again acutely neurotic.<br>Age 14: He continues to show his neurotic pattern of behavior. | In service 5 months, duty soldier. Medical discharge, not in line of duty, psychoneurosis not resulting from combat, manifested by anxiety reaction, chronic, based on latent homosexuality and by severe nervous tension, tremulousness, and occasional anxiety attacks. |

**Table 3.** ***Early psychiatric comments and subsequent service experience*— Continued**

| Case No. | Psychiatric comment | Experience in service |
|---|---|---|
| | *Unfavorable comment with unfavorable outcome* | |
| 8 | While he has made some progress, his problems are so set and of such long standing that he still isn't at a point where he can control himself and his feelings. At no time has he been accessible to real psychotherapy. | In service 16 months, no overseas duty, airman. Hospitalized eleven times for various physical difficulties and for psychoneurosis, hysteria. Medical discharge for psychoneurosis, anxiety. |
| 9 | We have enough information to indicate that he is an overt homosexual. There is apparently no, or little, feeling of guilt in connection with his homosexuality. I am of the impression that he is quite advanced in his homosexuality and does not wish help. | In service 54 months, Pacific theater 42 months. Twice reduced to Pvt.; highest grade reached, S/Sgt.; clerk-typist. AWOL once 9 and once 11 days. One summary court-martial for AWOL. Diagnosis at discharge: (1) Sexual deviate, overt homosexuality. (2) Psychoneurosis, chronic, severe, manifested by severe anxiety and depression, emotional instability, suicidal attempts. Incapacity severe. |
| 10 | The prognosis in this case appears to be very poor because of his instability and his marked hostility toward his father. It is not unlikely that in a rage he may commit violence. There is little that can be done about this, however, except perhaps to get him away from his present environment. | In service 10 months prior to World War II, bad conduct discharge for desertion; in service 3 months, 1943, with medical discharge, psychoneurosis. |
| 11 | It would seem that we are dealing with an emotionally unstable boy whose instability has been increased by a trauma to the central nervous system. His behavior warrants the provisional diagnosis of traumatic encephalitis. The possibility of a developing psychosis must be considered. | In service 36 months, no overseas service. Pfc., duty soldier, one reduction (Pfc. to Pvt.), 1 day AWOL. Medical discharge, psychoneurosis, manifested by nervousness, emotional instability, complaints of pain in back and difficulty in hearing and seeing, without adequate organic cause and with inability to perform duties. |
| | *Unfavorable comment with favorable outcome* | |
| 12 | He is a deeply neurotic boy with a great deal of aggression. | In service 35 months, 14 months in Europe. Sgt., administrative specialist, excellent character and efficiency ratings. |
| 13 | Age 10: He is a neurotic child with a marked need for attention, a need to create a sensation, and to punish others.<br><br>Age 12: It would seem well not to continue interviewing for the time being, despite the fact that he is neurotic and has conflicts which need solution. He has shown himself to be a rather uncooperative patient. | In service 48 months. Electrician's mate first class. |
| 14 | The contact with him was good and I am under the impression that therapy for his developing neurosis would be a possibility as far as the relationship is concerned. | In service 29 months, 20 months Korea. Sgt., radio operator. No disciplinary record or history of psychiatric contact. |

**Table 3.** ***Early psychiatric comments and subsequent service experience*—Continued**

| Case No. | Psychiatric comment | Experience in service |
|---|---|---|
| | *Favorable comment with favorable outcome* | |
| 15 | The examination of this boy reveals as normal a chap as the examiner has seen. He apparently is free from all conflict and does not display any objectionable behavior. | In service 40 months, in Europe 16 months; staff sergeant, platoon sergeant; Bronze Star Medal for action in France and Germany; superior efficiency rating. |
| 16 | Age 11: He seems reasonably stable except for the emotional display during the discussion on school.<br>Age 16: I feel that he is going to continue to get along well. | In service 43 months, 27 months in India, Sgt., radar operator and radar repairman. Excellent character and efficiency ratings. |
| 17 | This boy seems to be rather unusually well adjusted. He is well poised and gives no marked evidence of emotional tension. If anything, he is rather too well controlled and possibly lacking in the usual boyish vivacity and spontaneity. | In service 62 months, in Europe 25 months. Capt., special services officer, excellent and superior ratings. |
| 18 | He seems to be under no emotional difficulty and should make an easy adjustment at the training school. | In service 58 months, Pacific 9 months. Sgt., machine gunner and gun commander; expert infantry badge. Excellent character and efficiency ratings. |
| 19 | He is making a good adjustment, not only as his speech improves, but as he is capable of aggressive behavior. | In service 39 months, China-Burma-India 18 months. S/Sgt., airplane and engine mechanic and maintenance technician. Excellent character and efficiency ratings. |
| 20 | I am of the impression that he has a good hold on himself and that his de.inquency is under control. I think that he is going to get along very well. | In service 54 months; 31 months in Panama. 1st Lt., depot supply officer, efficiency reports excellent. |
| 21 | He has normal interests and adjusts well with other children. He is pleasant and friendly and is fully accessible. He does not exhibit any very unusual behavior. | In service 45 months. S/Sgt., veterinary technician; excellent character and efficiency ratings. |
| 22 | The problem does not appear to be a serious one. Mother was assured that she had no reason to be worried about him. | In service 43 months, 23 months in Pacific. Sgt., auto serviceman. Excellent character and efficiency ratings. |

In the earlier sample there were only 13 out of 110 cases with definite psychiatric comments; in the present sample there are 22 out of 98. For the two samples combined, these 35 cases can be classified into three groups:

1. Unfavorable comment with unfavorable outcome, 19 cases.
2. Unfavorable comment with favorable outcome, 5 cases.
3. Favorable comment with favorable outcome, 11 cases.

There were no cases with favorable comment and unfavorable outcome.

## SUMMARY

This is a replication of an earlier study which had indicated that a history of earlier peer-group difficulties tends to identify subsequent neurotic deviates in service. It has been found that a predictive procedure described before based primarily on information relating to earlier peer-group adjustment was equally effective with a new group of 98 individuals who, during childhood or adolescence, had been treated in a child guidance clinic. The 98 persons consisted of 49 who were diagnosed as psychoneurotic while in service and 49 who attained a grade of sergeant or higher without any adverse indications in their service records.

A second phase of this report concerns the relationship between psychiatric comments made in the course of the clinic treatment and subsequent outcome in service. It was pointed out that some types of comment were not discriminative with respect to subsequent service adjustment, but it was found that for the minority of cases where clear-cut diagnostic evaluations had been made, the long-time predictability was substantial. A detailed comparison of these predictions with later outcome is presented.


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## MEDICAL SEMANTICS

> The idea that a long word is more accurate seems to be inborn. For example I am sure you rarely use that accurate and short word "stroke." No, you write down cerebral thrombosis, cerebral hemorrhage, or cerebral embolus, according to your fancy, though you have little idea how to tell one from the other; even at post-mortem pathologists may be uncertain. Though there is this short word which covers all three, you choose a long word which describes a guess. It is odd. I suppose it is the dislike of sharing words with the laity.—RICHARD ASHER: Making Sense. *The Lancet*, September 19, 1959.

# Glutethimide (Doriden) Therapy and Overdosage

## A Review with Report of a Case

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GLUTETHIMIDE (DORIDEN), the sixth most frequently prescribed sedative hypnotic in this country,[1] is one of a group of anticonvulsant dioxopiperidine derivatives. It is absorbed with great variation. Fifteen hours after ingestion of normal doses of $C_{14}$-tagged glutethimide, 40 percent is excreted, 34 percent remains in the intestine, and 0.1 percent is in the brain and cord.[2] Whole tablets of the drug have been recovered from the stomach at autopsy 67 hours after ingestion of high doses. This may be related to its insolubility or to an "intestinal anesthesia." Absorption appears to be enhanced by alcohol.[2] Eighty-five percent of ingested glutethimide can be recovered from a biliary fistula.[2] It is reabsorbed by the intestine and excreted by the kidney.

After the ingestion of 1 gram of glutethimide, 1 to 3 mg per hour is excreted in the urine for as long as 60 hours. Ten to 30 percent is excreted as free alpha-phenylglutarimide; none is excreted unchanged. In cases of overdosage, a red-tan precipitate has often been reported in the urine after standing 24 hours.

Narcosis develops in the human when blood levels reach 2 to 3 mg per 100 cc.[1-3] Coma of 2 to 3 days has followed ingestion of 5 grams.[1] Fatal dosage has been as low as 10 grams, especially when alcohol or other depressants have also been ingested.[2, 4]

Habituation has recently been reported,[5-7] and reference is made to seizures during withdrawal of the drug.[5, 6] In animals no toxicity has been observed after several months of continuous use in normal dosage.[8]

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## OVERDOSAGE AND ITS THERAPY

There have been reports of at least 27 cases of serious intoxication from glutethimide with 7 fatalities.[1-3, 9-34] The course of intoxication is progressive, beginning with lethargy and confusion, and continuing through garbled speech, flushed face, coma, total flaccid paralysis, areflexia, and respiratory and circulatory collapse. In fatal cases necropsy has characteristically revealed cerebral edema, perivascular hemorrhage, hemorrhage of the galea, and pulmonary congestion. One case displayed massive adrenal infarction.

No characteristic laboratory findings are consistently noted. Spinal fluid pressure has been reported as nearly normal. Blood carbon dioxide concentration tends to be high and a high blood urea nitrogen was reported in a fatal case. Normal blood sugars have been noted. A rising white blood cell count often accompanies prolonged coma. Blood levels as high as 6.8 mg per 100 cc of glutethimide have been seen in a nonfatal case.

Typically, in addition to coma, paralysis, and varying levels of hyperreflexia, there is tachypnea, with 30 to 40 shallow respirations per minute. Abundant tenacious bronchial secretions are the rule, and pulmonary congestion, pneumonia, and hyperpyrexia often follow. Diligent tracheobronchial suction, maintenance of an airway (often tracheostomy is required), and oxygen therapy are essential. Circulatory collapse and persistent tachycardia are characteristic in more severe cases. Amphetamine and levarterenol have been effectively used for support of the blood pressure. Effective gastric lavage is an early requirement. Urethral catheterization with an hourly record of output is advisable. Fluid intake should probably be limited to 1,500 cc daily to prevent cerebral edema. Electrolyte intake should replace estimated losses only. Prophylactic antibiotic therapy should be considered.

A peculiar tendency to lapse into coma after initial response to therapy is a feature of many cases and may perhaps be due to renewed intestinal activity with concurrent absorption of remaining glutethimide. Coma frequently lasts from 36 to 72 hours in nonfatal cases. During manipulations of the patient, apnea frequently occurs. At least 1 case of cardiac arrest is described.

The use of bemegride as the stimulant of choice has been advocated, although the drug is now known not to be a metabolic antagonist of glutethimide.[2, 9] Bemegride is administered intravenously in 25 to 50 mg doses at 15-minute intervals until some return of reflexes or improvement of vital signs is noted. No attempt should be made to

administer sufficient drug to arouse the patient. If, as in the case reported below, bemegride is unavailable, caffeine sodium benzoate may be used cautiously with similar effect, in doses of 0.5 grams every 4 hours for 4 doses.

If no improvement is observed after 800 to 1,200 mg of bemegride has been given, or if convulsions occur, or if the condition is steadily deteriorating, the use of an artificial kidney is strongly recommended. Glutethimide is removed from the blood by the artificial kidney 100 to 400 times more rapidly than by the human kidney. In one case with an initial blood level of glutethimide of 3.1 mg per 100 cc, over 3 grams were removed during the first 4 hours of use of the kidney, but the rate of recovery thereafter fell rapidly.

## CASE REPORT

A 19-year-old white sailor was discovered comatose and totally unresponsive to painful stimuli at 0530 hours, 10 February 1959. History revealed that he had ingested 11 grams of glutethimide at 0100 hours, but at 0230 hours had been easily awakened by a shipmate. On the same evening he had taken two alcoholic drinks, but no other drugs.

### First Day

*0900 hours.* Physical examination on admission revealed the following: height 5 feet 7 inches, weight 155 lb, respirations 50 per minute and deep, blood pressure 100/70 mm Hg, pulse 120 per minute, and temperature 100.6° F rectally. Head, heart, lungs normal; bowel sounds absent. The patient was comatose and completely flaccid. Pupillary light reflexes were minimal, corneal reflexes absent, deep tendon reflexes hypoactive, and there was minimal withdrawal response to painful stimuli.

Laboratory data: serum sugar 95 mg per 100 ml, sodium 135 mEq per l, potassium 5.5 mEq per l, carbon dioxide 30 mEq per l, chlorides 107 mEq per l, Hb 15.5 grams per 100 ml, hematocrit 47 per 100 ml, white blood cell 10,000 per cu mm, and urinalysis normal.

Gastric lavage was performed and continuous drainage instituted, a urinary retention catheter was inserted, and the patient was given 5 percent glucose infusion. Intravenous injection of 2 cc of caffeine sodium benzoate caused transient slight increase in tidal volume.

*1230 hours.* Blood pressure 110/70 mm Hg, pulse 110, respirations 32. Respiratory volume was 4,400 cc per minute. Urinary output had been 150 cc per hour. With the exception of bilateral minimal pupil and Achilles reflexes, all reflexes and responses had disappeared. The patient was placed in an oxygen tent.

*1615 hours.* Vital signs and respiratory volume were unchanged. No reflexes remained. The pupils were dilated and fixed, and urinary output was 50 cc per hour.

Because of the rapid deterioration of the clinical picture the patient was transferred to Bellevue Hospital for extraction of the drug by use of the artificial kidney.

*1930 hours.* Vital signs were unchanged. Dialysis was instituted. Some slight reflex withdrawal was noted when the skin was incised for insertion of vascular catheters. After 5 hours on dialysis, only transient return of pupillary light reflexes were noted. Determination of glutethimide serum levels could not be obtained from any laboratory in the area. Prophylactic antibiotic therapy was instituted.

### Second Day

Vital signs were stable. Coma and areflexia continued, and consolidation of the left lung developed with a temperature of 104° F. A brick-red precipitate was noted in the urine voided the previous day.

*1900 hours.* Continuous peritoneal lavage was instituted. The patient displayed reflex withdrawal response and deepened respiratory excursion when the abdominal incisions were made, but the surgeon noted adequate muscular relaxation.

*2000 hours.* Bronchoscopy elicited major movements and cough response; copious amounts of dark, tenacious sputum were removed. The patient responded very little to less forceful stimuli and remained comatose.

### Third Day

Still on peritoneal lavage, the patient sluggishly opened his eyes. He made steady improvement and by 0700 hours was able to speak. Peritoneal lavage was discontinued.

### Fourth Day

Physical examination was entirely normal, as were chest roentgenogram, hemogram, and urinalysis. The patient was active, in good spirits, and was discharged ambulatory from Bellevue to St. Albans, where his convalescence was uncomplicated.

## SUMMARY

The course of intoxication following overdosage with glutethimide has been described, and recommendations for therapy are made. After a review of reports of serious intoxication appearing in the literature, a nonfatal case of intoxication after ingestion of 11 grams of glutethimide is presented. In large doses the drug has a long, often unpredictable period of absorption and excretion. Intoxication from overdosage, although occurring relatively infrequently, may be difficult to treat. In cases of overdosage, if any deterioration of the patient's condition develops, his management should probably be undertaken at an institution equipped with an artificial kidney.

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## HOW TO BECOME AN ALCOHOLIC

Since the alcoholic drinks to experience a sense of physical and emotional well-being, he soon discovers he must drink a lot more than he did a few years before because it takes more liquor to get the same "glow." So he begins to drink everyone under the table, and his friends often observe how well he can "hold his liquor." Thus his consumption increases markedly. So do his periods of temporary amnesia. Dubbed "blackouts" or "pulling blacks," these are not periods of physical unconsciousness. They are drinking episodes during which the developing alcoholic appears to an observer much like any other relatively drunk person. The difference, however, is the inability of the early-stage alcoholic to remember what happened during the drinking episode. At first these blackouts are sporadic, but in a few years they become regular experiences.—Harrison M. Trice: What We Know About Alcoholism in Industry. *Public Health Reports*, February 1959.

# Ligamentous Disruption of the Knee

## A Review with Analysis of 28 Cases

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TRAUMATIC DISRUPTION of knee ligaments is a disabling event resulting in an unstable knee that becomes a lifelong handicap in weight bearing. In the military service with its rigorous weight-bearing requirements, the rate of separation from the service because of chronic knee instability is unnecessarily high. It is believed that with this incapacity, only those military persons performing very limited and sedentary duties are fit for continued military service. The recurrent "giving way" of the unstable knee is a painful and disabling event that occurs with a frequency directly related to weight-bearing physical activity. Each episode results in pain, effusion of the knee, and further insult to articular surfaces and soft tissue support. The end result of continued knee instability is progressive, irreversible, degenerative arthritis.

It is well known that the results of late reconstruction of ligaments are less than satisfactory,[1] and it has been thought that failures to obtain stability were probably due to technical errors or absorption of the transposed material. Fascia lata and tendons have been used as cruciate ligament substitutes with unsatisfactory end results. There is a good anatomic reason for the failure of these late reconstruction attempts no matter what the technic or variations. A ligament is primarily a joint-stabilizing mechanism, and cruciate ligaments are characterized by a unique anatomic configuration. The functional role of ligaments is different from that of tendons and fascia, which resist or transmit linear tensile forces only. Fascia and tendon fibers are arranged in parallel fashion, those of tendons lying as a simple bound cable. On the other hand, a cruciate ligament, whose role is to provide joint stability in all ranges of motion, is composed of fibers that decussate freely to allow some to be taut at

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any joint position. Substitution of a tendon or fascial strip with its parallel fibers in the bed of the anterior cruciate ligament results in a knee that is stable in full extension, but progressively unstable as flexion increases. This repair, of course, offers the patient little benefit because the quadriceps sleeve anteriorly, and the hamstring tendons posterolaterally and posteromedially, supply sufficient stabilizing force for the knee in extension. On the other hand, with the knee in slight flexion, this sleeve-muscle mechanism is inoperative and the knee then depends upon its ligaments for support. Substitution of tissue for cruciate ligaments is thus doomed to failure.

The person with the unstable knee can be taught to assume a gait of full extension in weight bearing and cautioned to avoid unguarded weight-bearing movements, strenuous weight-bearing activity, and uneven surfaces in walking, and the thigh musculature can be strengthened. But it is only restoration of ligamentous continuity by early reparative surgery that can assure the patient a functional knee for the remainder of his life. One can anticipate almost uniformly successful and gratifying results if this is accomplished, as has been reported [2, 3] in a series of athletic injuries. One can certainly offer a gloomy prognosis if the ligaments are neglected. The reconstitution of this tissue is just as important as the repair of any other surgically accessible tissue in the body.

## ANATOMY

The ligamentous support of the knee is composed primarily of the medial and lateral collateral, and the anterior and posterior cruciate, ligaments. The anatomy and role of each ligament in stabilization of the knee has been well studied and reported.[4, 5, 6] An accurate knowledge of this anatomy, however, is essential for operative repair, as well as for understanding the pathomechanics of the injury and for meaningful examination of the injured knee.

The cruciate ligaments lie in the intercondylar notch and stabilize the knee primarily in the anteroposterior direction, although torsional and angular force is resisted by their obliquity as well. The ligaments themselves are surrounded by fatty synovial tissue and form an almost complete ligamentous mass in the intercondylar notch. They are decussating ligaments, the most anterior fibers at origin decussating to the most posterior portion of the ligament at its insertion. These crossed ligaments and the decussating intrinsic arrangement allow for stability in all ranges of motion.

The collateral ligaments primarily stabilize the knee against

angular force. These ligaments do not have the same intrinsic decussating fiber pattern, but the medial collateral ligament has its posterior portion composed of oblique interdigitating fibers that perform the same function as decussation in stabilizing the knee in all positions of flexion or extension. The fibers represent essentially a reinforced thickening of the quadriceps fascial sleeve and capsule, taut in full extension, slightly relaxed in flexion. The lateral collateral ligament arises from the lateral femoral condyle, lies on the surface although free from the joint capsule, and is intimately related to the lower fascia lata distally. The medial collateral ligament has a similar origin and insertion, intimately associated with the lower insertion of the thigh adductors; but it is worthy of note, surgically, that the insertion of this ligament lies some 3 inches below the joint line. This ligament actually passes beneath the medial hamstring tendons as they insert on the tibia at the junction of its metaphysis and shaft and passes deep to these tendons to insert distally on the tibial shaft. In addition, both medially and laterally there is a deep layer of collateral ligament. This short ligament arises from the same site proximally, is intimately associated with the capsule forming a sturdy base of attachment for the menisci, and inserts along the plateau rim of the tibia with the capsule.

## CLINICAL FEATURES

The force required to rupture the continuity of these ligaments is, of course, considerable. Most ruptures occur not from the usual twisting injury, but from forceful contact such as occurs in certain sports, or such as the blow of a moving vehicle against the leg when the foot is fixed upon the ground. Not infrequently, ruptures are caused by falls downstairs or from heights with a twisting angular force applied through the knee. The resultant pathologic triad is well known. With force applied to the knee from the lateral side, the usual result is a tear of the medial collateral and anterior cruciate ligaments, injury to the medial meniscus, or compression fracture of the lateral tibial plateau if ligaments fail to separate. When force is applied to the inner aspect of the knee, the opposite structures are involved. The common effect of hyperextension of the knee from force applied directly from the front is rupture of the posterior cruciate ligament as gliding extension is checked by the anterior cruciate, and the joint is forced open like a book posteriorly.

Usually the patient can describe the mechanism and direction of the force that was applied to his knee, and this should provide a clue



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for location of the lesion. He will state that the force applied was severe and that he felt a tearing, "giving 'way" of the knee, with excruciating pain and immediate consciousness of instability on attempting to stand and bear weight. There is a distinctive tearing sensation as opposed to the snapping, painful "pop" of the isolated damaged meniscus. Roentgenograms are not essential and usually are not helpful, although they may be useful in ruling out plateau fractures on the opposite side, and in serving as visual evidence of the instability, which might be indicated in some medicolegal situations. If the knee is seen within 30 minutes of injury, effusion, swelling, and pain on motion are usually not severe enough to prevent examination for ligamentous integrity. In the ensuing 2 hours, however, the picture is altered by the presence of bleeding within or about the joint, protective muscle spasm, and apprehension on the part of the patient. Effusion is not generally a feature of this injury, inasmuch as a tear of a collateral ligament always results in laceration of the synovium with resultant escape of joint fluid. In the isolated cruciate tear, however, as seen in hyperextension injury and tear of the posterior cruciate ligament, there may be brisk bleeding and considerable tension within the joint.

If the collateral ligaments are involved, tenderness can be elicited with almost pinpoint accuracy at the site of rupture. The most common injury involves the medial collateral ligament, and this ligament is most commonly torn from its tibial attachment distally so that this is the point of maximum tenderness. Usually tenderness is elicited medially in the joint line, indicating disruption of the deep layer of this same collateral ligament. Full extension of the knee may be obtained by gentle manipulation unless an associated locked meniscus is present. When the patient is seen perhaps 12 or 24 hours later, ecchymosis is usually present distally along the flare and shaft of the proximal portion of the tibia. The points of tenderness are discrete and the joint is free of effusion.

The usual method of testing the stability of the collateral ligaments is satisfactory for diagnosis (fig. 1). Gentle opening of the extended knee with careful palpation of the joint space as gentle pressure is applied will reveal abnormal opening of the joint space. The feeling is characteristic and the examination can be done at any time following injury if done in a nontraumatic manner. Forceful manipulation is neither warranted nor necessary. Testing for cruciate stability by the method of drawing the tibia forward and pushing it backwards on the femur with the knee in flexion, the so-called "drawer" sign or manipulation, is unsatisfactory. It is a painful procedure and is

resisted by the patient with muscle spasm; with the knee flexed at approximately 90°, tension of the spastic protective hamstrings will lead to false findings and frequently to a false negative test. The hamstring tendons can easily overpower manual attempts at sliding the tibia forward on the femur. Cruciate testing is best performed with the knee in full extension, at which position the hamstrings or quadriceps cannot resist the examiner by contraction.

**Figure 1. Test for integrity of medial collateral ligament. The joint space is opened beneath the palpating index finger.**

To test the anterior cruciate, the knee is extended in a comfortable position (fig. 2). The palpating index finger is placed in the small triangle formed by the curved femoral condyle and straight tibial plateau medially. The palm of this same hand stabilizes the femur while the second hand gently lifts the tibial plateau anteriorly. If instability is present, the tibial plateau can be easily palpated sliding forward under the palpating finger.

**Figure 2. Anterior cruciate testing. The tibial plateau is lifted anteriorly.**

To establish the integrity of the posterior cruciate, the maneuver and hands are reversed (fig. 3). The examining finger of the second hand is placed in this same triangle between femoral condyle and tibial plateau, the palm stabilizing the tibia. Then, the first hand, placed beneath the femoral condyles, gently draws them forward, the motion being easily palpated by the examining finger. Hyperextension of the knee indicates loss of continuity of the posterior cruciate (fig. 4). The uninjured knee must be examined for comparison.

**Figure 3. Posterior cruciate testing. The femoral condyles are lifted anteriorly.**

**Figure 4. Hyperextension indicates posterior cruciate disruption.**

An accurate diagnosis of what structures are damaged and the extent of damage must be made, and this cannot be deferred during

trial of conservative therapy in hopes that later reconstruction, if necessary, will result in a serviceable knee. With gentleness and care, the diagnosis can be made in practically all cases. If the patient's discomfort and apprehension preclude examination, the physician is justified in examining the damaged knee under anesthesia, as the future of this extremity in weight bearing depends upon early accurate diagnosis. Once the diagnosis of complete disruption of one or more ligaments of a previously stable or normal knee is made, surgical repair is indicated unless age or other disabilities contraindicate, or unless the occupation of the patient does not require a strong stable knee. Incomplete tears present the symptoms and signs of a complete tear, that is, pain, tenderness, and ecchymosis, but do not demonstrate instability on the tests described above. These incomplete tears or "sprains" are best treated by immobilization in a long leg plaster cast, with the knee flexed between 135° and 150° to allow healing of the ligament in the relaxed position.

## TREATMENT

Surgical repair must be performed promptly following the diagnosis of ligamentous disruption. However, this is never an emergency procedure, and time should be taken for thorough skin preparation and thorough evaluation of the patient. The operation can then be scheduled in 24 to 48 hours. A longer wait is justified if surgical incisions would otherwise be made over abraded contaminated skin. The operation itself is performed under regional or general anesthesia, with tourniquet control and with adequate operating room facilities and personnel.

**Figure 5. The parapatellar incision, extended distally and curved posteriorly to expose collateral and cruciate ligaments.**

If the medial aspect of the joint has been damaged, a long median parapatellar incision is used, extending distally and curving posteriorly at the junction of the metaphyseal flare and shaft, so that both collateral and cruciate ligaments can be approached and repaired as the injury indicates (fig. 5). After the medial skin flap has been reflected, the superficial layer of the collateral ligament may be inspected with manipulative opening of the medial joint space. As indicated earlier, the site of disruption is usually found distally and may be completely hidden by the hamstring tendons unless these are retracted and the ligament traced to its insertion. The superficial laceration is opened widely by extending the incision transversely and proximally toward the patellar tendon to permit a median parapatellar exposure of the joint space itself. This superficial ligament layer is reflected proximally and medially and the status of the deep layer is evaluated.

If the deep layer is torn from the tibia, inspection of the meniscus and cruciate ligaments is simple. When cruciate damage is present, the incision, as mentioned, is extended proximally in the median parapatellar route and the patella dislocated laterally to expose the intercondylar notch. The knee is then placed in flexion and meniscectomy and repair of the cruciate ligaments is accomplished as indicated.

If the anterior cruciate is detached from its anterior bony insertion, it is simply resutured by pulling the ligament back to its bony bed with a suture passed through a drill hole and tied superficially in the soft tissue below the joint level. This draws the cruciate back to its normal insertion, where it can be expected to heal. If the posterior insertion is avulsed, a drill hole is passed postero-transversely through the lateral femoral condyle from its bed of origin, the bone is freshened, and a simple pull-through suture is tied over a small buttress of gauze on the skin posterolaterally, thus holding this ligamentous origin in place for reattachment.

The anterior attachment of the posterior cruciate is similarly treated, if necessary. The posterior tibial insertion, however, is quite difficult unless anterior cruciate avulsion allows visualization. The bony bed is palpated behind the anterior cruciate and freshened with a curet. The ligament is pulled to it by a suture, passing a blunt, long tendon passer posterolaterally through the popliteal space over the bony bed to draw the ligament into place.

Disruption of these ligaments in continuity requires simple suture only. This is accomplished with the knee bent at about 135° to 150°, and then this portion of the incision is closed. The deep and super-

ficial layers of the collateral ligament are next repaired by simple suture in their normal position and with normal suture tension. The meniscus is not removed unless torn from its base on the deep layer of the collateral ligament and capsule.

The knee is maintained in a long leg plaster cast with the knee flexed at 135° to 150° postoperatively for 21 days, at which time healing is considered to be adequate for gentle motion. At this time, supervised motion is started in flexion only, utilizing the cast shell for protection and to prevent extension. Six weeks after surgery extension from this position is initiated. Extension is delayed for this period because it produces tension upon these repaired ligaments. Rehabilitation is slow and the average time to regain full extension with recovery of quadriceps power to normal is about 16 weeks. Return to full military duty should be delayed until these criteria are met.

## ANALYSIS OF CASES

Twenty-eight consecutive cases treated by the author between 1947 and 1958 are presented. Most of these patients were young active men between 20 and 28 years of age, except for one 42-year-old patient. All injuries were the result of severe force, 75 percent having been incurred as athletic injuries, and the remainder as the result of twisting force in falls usually from a considerable height. Only one was incurred in an automobile accident.

Analysis of the operative findings indicates the following distribution of ligament damage:

| | |
|---|---|
| Anterior cruciate only (torn menisci 6) | 11 |
| Anterior cruciate, and medial collateral (torn meniscus 1) | 7 |
| Medial collateral only | 4 |
| Anterior cruciate, posterior cruciate, and medial collateral | 4 |
| Posterior cruciate and lateral collateral | 1 |
| Posterior cruciate only | 1 |
| Total | 28 |

A torn medial meniscus requiring removal of the fragment was present in only 5 cases. In two additional cases, both meniscuses were torn. All meniscus tears were associated with an isolated anterior cruciate tear except one, and this involved a tear of both anterior cruciate and medial collateral ligaments. There was no instance of tear of the meniscus from its attachment when only the medial collateral ligament had been disrupted. Five cases of isolated anterior

cruciate tear were not complicated by tear of the meniscus. This suggests that abnormal anterior displacement of the tibia on the femur is the mechanism of tear of the meniscus, rather than forceful opening of the medial joint space.

One complication occurred in the form of a severe postoperative wound infection involving the joint, which required surgical drainage and prolonged antibiotic administration. This patient was separated from the service because of limitation of motion and scarring about the knee. The knee, however, was stable.

All other patients returned to full military service with stable knees and normal ranges of motion, all having full powerful extension and flexion past 90°.

## SUMMARY

The anatomy of the knee ligaments and the clinical features of disruption of these ligaments are reviewed. Surgical repair of ligamentous disruptions is described, and an analysis of 28 operated cases is presented.

A plea is made for the early surgical repair of disrupted knee ligaments. The results of delayed reconstructive procedures are disappointing, and it is only by early diagnosis and early surgical repair that a stable, functional knee can be assured.

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# The Headache Problem

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HEADACHE is one of the most frequent complaints confronting the clinician whatever his specialty. It has been estimated that 50 percent of patients encountered in general practice suffer from headache.[1] In an unselected group of 10,000 men between the ages of 18 and 38 who were examined for military service during World War II, 8 percent complained of severe headache.[2] In a large industrial plant, an investigation into the medical reasons for 15,000 absences among employees revealed that 24 percent were due to headache.[3] Tunis and Wolff [4] claim that 70 percent of the population have headache at one time or another.

Yet despite its frequency and importance, the headache problem has been neglected. It may receive little attention in the medical school curriculum, is seldom the topic of interest of more than a few doctors, and too often is left to some one else to worry about—usually the suffering patient. And with neglect have come carelessness in diagnosis, misconceptions about etiology, errors in treatment, and dissatisfied patients who at times seem destined to be inflicted forever with the scourge of headache.

It is the attempt of this review to outline the subject of headache, to dispel some of the misconceptions associated with it, and to offer a simple, working classification of headaches together with their characteristics, mechanisms, and treatment.

## MISCONCEPTIONS

Misconceptions concerning the causes of headaches are almost as common within the medical profession as among the lay population. Contrary to popular belief, headache can rarely be attributed directly to diseases of the eyes, ears, paranasal sinuses, nose, or teeth. Even when abnormalities are discovered in one of these areas and are remedied, the patient may find his headache unabated.

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Many also describe a causal relationship between headache and constipation, menstruation, pregnancy, the menopause, vitamin deficiency, allergy, and hormone imbalance. Yet seldom do these factors have any direct etiologic significance. Neither is there any direct relationship between elevated cerebrospinal fluid pressure and headache. It has been demonstrated that the experimental increase of cerebrospinal fluid pressure to over 500 mm of water does not produce headache in the normal subject.[2] Again headache has been produced on the side of the lesion in a patient with brain tumor by lowering the intracranial pressure but not by elevating it to 550 mm.[5]

## CLASSIFICATION OF HEADACHE

Basically, headache depends upon the stimulation of extracranial or intracranial pain-sensitive structures. All tissues, especially the arteries, overlying the cranium are, to a greater or lesser extent, sensitive to pain. In addition, the various tissues about the face and neck (that is, the eyes and ears and their component parts, the nose, paranasal sinuses, et cetera) are pain sensitive, and may occasionally, when stimulated, be a cause of cephalgia.

The intracranial pain-sensitive structures include the great venous sinuses and their venous tributaries from the surface of the brain, parts of the basal dura, the dural arteries and cerebral arteries at the base of the brain, and the fifth, ninth, and tenth cranial and upper 3 cervical nerves.

The mechanisms of headache of extracranial etiology are numerous and diverse. The vascular headaches result from an inflammatory process, such as cranial arteritis, or from extracranial vascular dilatation, as in migraine or histaminic cephalgia. Tension headache is probably due to autonomic nervous system stimulation. Those rare headaches which are caused by localized disease of a specific extracranial structure such as the eyes, ears, or teeth may have any one of a number of mechanisms—inflammation, direct compression, direct stimulation of a nerve ending or muscular strain and spasm.

However, the basic mechanisms of intracranial headache are limited in number. Wolff [2] considers six: (1) traction on veins that pass to venous sinuses from the surface of the brain, and displacement of the great venous sinuses; (2) traction on the middle meningeal arteries; (3) traction on the large arteries at the base of the brain and their main branches; (4) distention and dilatation of the intracranial arteries; (5) inflammation in and about the pain-sensitive structures; and (6) direct pressure on the cranial and cervical nerves containing afferent fibers from the head.

On the basis of the pain-sensitive structures and mechanisms, one can formulate the following classification of headache:

I. HEADACHE OF EXTRACRANIAL ETIOLOGY
- A. Nonvascular Headache
  1. Sustained muscular contraction headache (tension headache)
  2. Disease of the eyes
  3. Disease of the nose, ears, and paranasal sinuses
  4. Disease of the teeth and gums
  5. Disease in and about the neck
- B. Vascular Headache
  1. Disease of the superficial cranial arteries (cranial arteritis)
  2. Fever, foreign protein reaction, and reactions to bacterial and other toxins — Due to extracranial vasodilatation
  3. Histaminic headache — Respond to ergot derivatives — Due to extracranial vasodilatation
  4. Hypertensive headache — Respond to ergot derivatives — Due to extracranial vasodilatation
  5. Migraine headache — Respond to ergot derivatives — Due to extracranial vasodilatation

II. HEADACHE OF INTRACRANIAL ETIOLOGY
1. Inflammatory lesions
2. Space-occupying lesions
3. Organic intracranial vascular lesions
4. Cerebrovascular accidents

III. MISCELLANEOUS HEADACHES
1. Post-traumatic headache
2. Post lumbar puncture headache
3. Postpneumoencephalography and postventriculography headache
4. Dehydration headache

## CLINICAL FEATURES

*Tension headache.* Tension headache is by far the most common type of headache seen in the general practice of medicine and alone probably accounts for 90 percent or more of all headaches.

In contrast to migraine headache, prodromata are characteristically absent. The pain typically involves the frontal and occipital regions but may involve the entire head in a caplike distribution. It is usually described as pressure-like viselike, or resembling a tight hatband and is invariably associated with a sensation of tightness or spasm of the posterior neck and shoulder muscles. Frequently, there will be palpable spasm or even tender nodules within the musculature of the head, neck, and upper back. There may be associated anxiety, nausea, and vomiting, the latter two features frequently being responsible for the incorrect diagnosis of migraine. The frequency and duration of the headache is variable but rarely lasts less than 1 hour, often persists for an entire day to 3 or 4 days, and may last for several weeks.

Friedman, von Storch and Merritt[6] reviewed 1,000 cases of tension headache, of which 65 percent were female and 40 percent had

a family history of headache. In 55 percent headache developed between the ages of 20 and 40. In 20 percent there was complaint of constant headache with no freedom from pain. Thirty percent of patients stated they had daily headache and only 15 percent had headache less than once each week.

The physiologic mechanism of tension headache is not well understood. Apparently mental conflict produces autonomic nervous system stimulation, which in turn causes stimulation of the somatic motor system with resultant painful persistent contraction of muscles of the head and neck. However, some feel that tension headache is due to painful vasodilatation which follows early initial vasoconstriction.[7, 8]

The cause of tension headache is likewise problematic, but, as the name implies, emotional factors are probably of primary etiologic importance in 100 percent of cases. While the underlying emotional conflicts are usually unconscious, many patients relate the onset of each headache to some psychic upset or will admit to feeling emotionally upset without any apparent reason prior to the onset of cephalgia. This author's experience indicates a remarkably high incidence of emotional depression and hyperventilation syndrome in patients with tension headache. Fully 40 percent of patients seen have freely admitted to feelings of depression, or to symptoms suggestive of depression, such as marked morning fatigue despite an apparent good night's rest, deep sighing respirations, restlessness, loss of ambition and interests, frequent unexplained desire to cry, or actual overt crying. An almost equal number of patients suffer from symptoms of hyperventilation, including sudden onset of lightheadedness, visual blurring, paresthesias of the toes, fingers, scalp, and circumoral region and sometimes syncope. So frequently have these three features—depression, hyperventilation, and tension headache—been associated that any patient confronting a doctor with one of these complaints will generally have the other two.

Friedman, von Storch and Merritt [6] found that the most frequent sources of conflict in tension headache are hostile impulses, with hostility, resentment, and aggression being directed against family members or family surrogates. Intellectual, social, and financial demands beyond the individual's ability may initiate headache. It may develop when the subject has reached his tolerance of suppressed anger and resentment. Some develop headache as a source of gaining attention or as a method of identification with important, usually family, figures.

In treating the patient with tension headache, one can expect little success from drug therapy alone. Unquestionably, psychotherapy is

mandatory and of primary importance if the patient is ever to be freed of headache. The single most important feature in the successful handling of any headache patient is the establishment of a satisfactory doctor-patient relationship. Not until the doctor has won the patient's complete confidence and revealed himself to be an interested, understanding and sympathetic observer will the patient permit exploration into the conscious and unconscious factors responsible for his symptom.

An initial detailed history and complete physical and neurologic evaluation are most important despite the obvious non-organic origin of the headache. The doctor's interest and thoroughness become immediately apparent and do much to reassure the patient as to the non-organic etiology of his head pain and to establish necessary confidence in the physician.

Next, it is of great value to educate the patient as regards his headache—discussing misconceptions, probable mechanisms, etiology, treatment, and the effects of emotional stress and conflicts on body physiology.

Once the necessary rapport is established, the patient is encouraged to ventilate and "talk out" his problems. If the emotional conflict stems from a realistic social problem, such as financial instability, unemployment, or incompatible marriage, a social worker or agency can be of invaluable aid. If the emotional conflict is too complex or severe for the nonspecialist to manage, a psychiatrist should be recommended. Whatever the form of psychotherapy, the primary treatment must be to aid the patient in evaluating and understanding his responses to his environment and his relationships with others with the hope of finding a way of coping with stressful situations.

The most effective medications in treating tension headache are combinations of an analgesic, a sedative, and caffeine.[9] Sedatives and analgesics when used alone are less effective. Reserpine, chlorpromazine hydrochloride, meprobamate, and Ultra (phenaglycodol) are said to be helpful.[10] This author has obtained good results with 5 mg of Dexedrine (dextro-amphetamine) sulfate, 50 mg of Amytal (amobarbital) sodium, and 600 mg of salicylate sodium, used in combination every 6 hours as required. Dexedrine and Amytal are also effective in combating depression and hyperventilation syndrome. Friedman, von Storch and Merritt [6] recommend the use of the sedative-analgesics Fiorinal and Daprisal. It is important to remember, however, in evaluating the beneficial effect of any drug in the treatment of tension headache, that relief is obtained in 50 percent or more of patients with the use of placebos.

*Headache due to disease of the eyes, ears, nose, sinuses and teeth.* Headache secondary to disease of the eyes is rare. Refractive errors may at times be responsible for head pain, but such discomfort is invariably clearly related to excessive use of the eyes and is felt in the immediate vicinity of the eyes. Frank abnormalities such as inflammatory lesions and glaucoma may produce severe eye and head pain, but signs of ophthalmic disease will usually be apparent. While it is agreed that ocular muscle strain can cause headache, the "post-movie headache" may be due as much to the emotional experience in seeing the movie as it is to strain of the ocular muscles.

Disease of the ears, nose, and paranasal sinuses may be a cause of headache but will almost always be accompanied by symptoms referable to these areas and signs of local pathologic change. However, it is important to remember that the frequent association of migraine headache with unilateral lacrimation, conjunctival injection, edema of the nasal mucosa, nasal stuffiness, and rhinorrhea may result in the false diagnosis of upper respiratory disease.

Headache of dental origin is not common. Wolff [2] has demonstrated that electric stimulation of a tooth will cause pain in the area of the tooth and produce homolateral headache of the face and temple, lasting a considerable time after cessation of the stimulus and after the local pain has disappeared. Headache may also appear as the result of erupting impacted third molars.[11] However, headache which results from dental disease is usually accompanied by pain in the tooth itself, leading to proper diagnosis.

*Headache due to disease in and about the neck.* Tightness of the posterior neck muscles is common in tension headache. However, primary disease of the neck and its associated structures, such as osteoarthritis, herniated disc, myositis and bruises, may initiate headache. Therefore, the neck should be examined carefully in all cases of headache.

*Headache due to cranial arteritis.* The headache of cranial arteritis is caused by direct inflammation of the involved vessels, usually the temporal or occipital arteries, with swelling, tenderness, and loss of pulsations of the involved vessels. Mental confusion and diminished auditory and visual acuity may be observed. Fever, elevated erythrocyte sedimentation rate, and leukocytosis are often present. Salicylate, steroid, and anticoagulant therapy may constitute sufficient treatment. Excision of the involved vessel usually results in cure and confirms the diagnosis.

*Headache due to fever, foreign protein, and toxins.* When histamine is injected intravenously into the normal subject, generalized

vascular dilatation and transient fall in blood pressure occur. Because the peripheral vasculature regains its normal tension and caliber before the intracranial vessels contract, the intracranial vessels are still dilated during the return of systolic force. This produces still greater dilatation and pulsation and results in severe throbbing headache. Headache caused by fever, infection, and the injection of foreign protein or toxins is said to have this same mechanism.[12, 13] Similar headaches result from exposure to low oxygen, as in carbon monoxide poisoning or sudden fall in blood pressure from any cause with resultant hypoxemia and painful, compensatory intracranial vascular dilatation. Because these headaches do not respond to vasoconstrictor drugs, relief depends mainly on analgesics.

*Histaminic headache.* Histaminic headache was first described by Horton [14, 15] and is variously referred to as "Horton's syndrome," "Horton's headache," "autonomic cephalgia," and "cluster headache." It is characterized by the explosive onset of severe, unilateral, throbbing, or burning pain in the temple, eye, forehead, cheek, or region behind the ear and upper part of the neck. It is often associated with homolateral tearing, miosis, enophthalmos and conjunctival injection, ipsilateral rhinorrhea and nasal obstruction, facial flushing and sweating, and occasional nausea and sialorrhea. It usually occurs in men after the age of 40 and at night, frequently rudely awakening the patient 1 or 2 hours after he has gone to sleep. It lasts from 15 minutes to several hours and may be of such severity as to cause crying and thoughts of suicide. These headaches often appear in clusters, occurring every day or night or several times a day or night for weeks, months, or years. Sometimes a patient never has a recurrence after a single cluster, or at the other extreme the period of freedom may gradually shorten until he is suffering from daily headache for years. An injection of histamine subcutaneously (0.35 mg) will often precipitate a classic headache during the time the patient is susceptible. However, generalized headache immediately following subcutaneous histamine and lasting 5 to 10 minutes should not be confused with a positive test for histaminic cephalgia. This initial headache is called a histamine headache and occurs even in the normal subject who receives histamine parenterally. The typical attack of histaminic cephalgia will occur 15 to 50 minutes after cessation of the histamine headache. A positive test is obtained in 60 percent of cases.

The mechanism appears to be localized dilatation of branches of the external carotid artery, and an attack is usually rapidly terminated by the administration of a vasoconstrictor drug such as epinephrine or one of the ergot derivatives. Antihistamines are seldom of benefit. Histamine desensitization to prevent future attacks is still considered

the treatment of choice by Horton.[15] After initial relief of the headache, an attempt may be made to get the patient out of a "cluster" by encouraging rest and the use of ergot two or three times daily. Psychotherapy, with the hope of relieving tension, should be instituted.

*Hypertensive headache.* There is some question as to whether hypertensive headache is a true entity. Some cardiologists feel that when headache occurs in hypertensive patients, it is usually coincidental and has its origin in any of the usual causes of headache. Tension headache may result in the hypertensive patient who is aware of the seriousness or potential seriousness of his illness and worried about its outcome. Wolff[2] feels that the headache of hypertensive patients bears no relationship to the level of the blood pressure and may occur when the blood pressure is low, moderate, or high. However, he believes that headache in the hypertensive patient may be due to further dilatation of vessels already dilated from any cause.

The "hypertensive" headache is usually described as a bilateral, dull throbbing in the nuchal and temporal regions, especially severe on awakening in the morning, and gradually improving after arising. Sitting up often prevents or minimizes the headache, whereas stooping over and coughing may increase its intensity. Wolff[2] describes a second variety of hypertensive headache which may occur in association with the above or independently and which is indistinguishable from typical tension headache.

Hypertensive headache should respond to ergot in the acute phase. Frequent or prolonged use of ergot preparations, however, is ordinarily contraindicated in hypertension. Prophylactic and definitive therapy consists of antihypertensive measures.

*Migraine headache.* Migraine is the second most common type of headache, and it is estimated that 2 to 8 million people in America are temporarily incapacitated by it.[16] It is characteristically paroxysmal, periodic, unilateral, and throbbing, and is usually preceded by visual and psychologic disturbances and accompanied by nausea, vomiting, and irritability. Friedman, von Storch and Merritt[6] in their analysis of 1,000 patients with migraine found that 65 percent had a positive family history, that 70 percent were females, that 55 percent developed headache before the age of 20, and that onset after the age of 40 was rare. Prodromata were present in 75 percent, and 80 percent claimed that the headache was unilateral. In women, migraine is frequently associated with menstruation and often tends to diminish after the menopause.[17] The ingestion of alcohol or chocolate may also initiate migraine.[17] Migraine is common in children, in whom the syndrome is no different than in adults except that abdominal symptoms may overshadow the headache.[6]

The typical patient, then, is a female who periodically develops transient irritability, photophobia, visual blurring, scintillating scotomata, or hemianopsia, followed by throbbing pain which may become a steady aching in one supraorbital-frontal or temporal region. The pain may become generalized throughout the head and at times is excruciatingly severe. Usually associated nausea, vomiting, and prostration occur. Frequently one observes, on the side of the headache, a distended temporal artery tender to palpation. Compression of the involved vessel or the common carotid artery on the side of the head pain often produces temporary relief.

Some patients have unilateral lacrimation, conjunctival injection, nasal obstruction, and rhinorrhea, with edema and injection of the nasal mucosa on the same side as the headache. Homolateral facial flushing may be noted. Migraine headache has also been described in association with unilateral oculomotor paralysis.[18] However, unilateral frontal headache in association with oculomotor paralysis should always suggest an aneurysm of the internal carotid artery.

Abnormal electroencephalographic patterns consistent with focal brain damage have been reported in migraine headache.[19, 20] The changes usually occur at the time of the prodromal, visual, or other neurologic phenomena and revert to normal when focal neurologic manifestations disappear and headache begins.

All or only a few of the above symptoms may occur with any particular migraine episode and usually last less than 1 day. Rapid relief invariably follows intramuscular 0.5 mg of ergotamine tartrate in migraine and in all vascular headaches except cranial arteritis and those due to fever, foreign protein, or toxins. When no headache is present and migraine is suspected, a typical headache may be induced with 1.2 mg of nitroglycerin sublingually.[17] A positive result is not to be confused with the immediate headache which is due to this drug. The typical headache occurs about 1 hour after the drug is given.

The migraine syndrome is not limited to the head but may manifest itself through other systems.[6] The most common migraine equivalent is "abdominal migraine," characterized by periodic abdominal pain, nausea, and vomiting and often accompanied by typical migraine headache. It is usually seen in patients who suffer from periodic migraine headache but may occur independently of cephalgia.

"Precordial migraine" has been described and presents as precordial pain radiating to the left axilla and arm. The migraine syndrome limited to the limbs, pelvis, or genitalia has been rarely reported.

The mechanism of migraine headache is well understood.[21] An initial vasoconstriction of branches of the internal carotid artery



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produces visual and other pre-headache phenomena. This is followed by dilatation and distention of branches of the external carotid artery, usually the superficial temporal branch, with stimulation of pain-sensitive nerves in and around the vessels. This may be followed or accompanied by contraction of head and neck muscles, with tension headache resulting either from direct stimulation of nerve endings or from ischemia of the muscles. Recent work [22] indicates that a local substance in migraine headache lowers pain threshold and damages tissue. When edema fluid, which accumulates about the dilated vessels at the area of headache is removed and analyzed, its amino acid content is found to be increased as the result of local protein breakdown. When injected intracutaneously into other areas, this edema fluid produces erythema and slight decrease in cutaneous pain threshold.

The cause of migraine headache is not known, but emotional factors are of great importance. Frequent mention is made of the "migraine personality"—a compulsive, meticulous, rigid, anxious, and intelligent person who is hypersensitive, over-conscientious, and a perfectionist, aggressive toward his environment, and resentful of his lot in life. However, there is no conclusive evidence that a distinct migraine personality pattern exists.

As in the treatment of tension headache, psychotherapy is of utmost importance in controlling the frequency of headaches. However, unlike drug therapy in tension headache, medications for combating migraine are successful and specific. They act as vasoconstrictors on branches of the external carotid arteries. The success of a drug in rapidly aborting an acute attack of migraine is enhanced if the patient takes it during the prodromal stage at the very first sign of the syndrome rather than awaiting the presence of actual head pain. The immediate treatment of migraine is said to be most effective with oral or rectal ergotamine tartrate and caffeine (Cafergot).[6] However, any of the following ergot derivatives may be used successfully:

1. Dihydroergotamine: 1 ml (1 mg) IV.
2. Dihydroergotamine: 1 ml (1 mg) SC.
3. Gynergen (ergotamine tartrate): 0.5 ml (0.25 mg) IM.
4. Cafergot tablets: 2 tablets followed by 1 tablet every 30 minutes until relief is obtained or until a total of 6 tablets have been taken.
5. Cafergot suppositories: 1 suppository followed by an additional one in 60 minutes if required.
6. Cafergot P–B tablets: 2 tablets followed by 1 tablet every 30 minutes until relief is obtained or until a total of 6 tablets have been taken.

7. Cafergot P–B suppositories: 1 suppository followed by an additional one in 60 minutes if required.

8. Ergotamine tablets: 2 tablets followed by 1 tablet every 30 minutes until relief is obtained or until a total of 6 tablets have been taken.

The suppositories have been most useful in cases where vomiting prevents retention of oral medication. Cafergot P–B contains pentobarbital sodium and an antispasmodic to combat nervous tension and gastrointestinal symptoms, respectively. Approximately 15 percent of patients are refractory to drug therapy and 35 percent to preventive measures, that is, psychotherapy.

Certain precautions should be taken in using ergot derivatives. The maximum dose of ergotamine is 10 mg in any one day and this should not be given more than once each week. Side effects of excessive ergotamine include nausea, vomiting, paresthesias of the hands and feet, pains in the neck, thighs, and abdomen, and substernal oppression. Muscle spasm of the extremities and perivascular pain may occur. The drug is contraindicated in organic heart disease, obliterative vascular disease, hypertension (except for occasional use in the acute phase), pregnancy, liver disease, and septic states associated with intravascular foci.

*Headache due to cranial inflammation.* Headache as a result of cranial inflammation is usually obvious. It is invariably due to direct stimulation of the sensitive areas of the meninges or arteries by the various agents which cause meningitis or by distortion of pain-sensitive structures from changes in cerebrospinal fluid dynamics. The usual signs and symptoms of meningitis are invariably present.

*Headache due to space-occupying lesions.* Intracranial neoplasms, abscesses, cysts, and hematomas are uncommon causes of headache. The mechanism is distortion of pain-sensitive structures by direct pressure on these structures or by spinal fluid pressure changes.

Because one should always rule out brain tumor as the underlying cause for any headache and because patients want reassurance that a neoplasm is not the cause, a detailed discussion of brain tumor headache is in order.

Headache is seldom the sole symptom of brain tumor. Other neurologic symptoms and signs are usually present, such as diplopia, hemiparesis, facial paralysis, ataxia, reflex changes, and convulsions. Wolff[2] states that when present, headache due to brain tumor is usually a deep, dull aching pain and seldom of a throbbing quality. It is intermittent, rarely intense, and usually relieved by aspirin or by the application of cold packs to the head. It may be increased in

intensity by coughing and straining at stool and may be worse in the erect than in the recumbent position.

Although headache as an aid in localizing the tumor is of limited value, it may help significantly in localization if the headache is continuous, as it is in 10 percent of cases. The following rules are summarized from Wolff:[2]

1. Headache overlies the tumor in about one third of all patients.
2. In two thirds of patients without papilledema, headache overlies the tumor.
3. In all patients without papilledema, unilateral headache is on the side of the tumor.
4. When headache is both frontal and occipital, it has little localizing value.
5. When headache is continuous, its localizing value is greatly increased.
6. Headache is almost always present in posterior fossa tumors, is usually the first symptom, and is usually occipital.
7. Headache may be absent in supratentorial tumors, is the first symptom in one third of cases, and is usually frontal in the absence of papilledema.
8. Headache from supratentorial tumors is rarely occipital unless associated with papilledema; and even when headache is occipital, there is usually frontal pain as well.
9. Headache of cerebellopontile angle tumors is often—and sometimes only—postauricular on the side of the tumor.

*Headache due to organic intracranial vascular lesions.* Hemangiomas, arteriovenous fistulae, and aneurysms may cause headache resulting from distortion and direct pressure on pain-sensitive structures, from vascular dilatation and painful stretching of the lesion itself, or from hemorrhage.

*Headache due to cerebrovascular accidents.* Although not a prominent feature of the cerebrovascular accident, headache may occur. When thrombosis or embolization causes the accident, headache probably results from distention and ischemia of the walls of the involved vessels and compensatory dilatation of neighboring arteries. Hemorrhage may cause severe pain for the same reasons or as a result of distortion of pain-sensitive structures by the expanding mass of blood.

*Post-traumatic headache.* Headache following trauma to the head and neck is difficult to evaluate. It may result from epidural, subdural, or subarachnoid hemorrhage or be due to localized trauma to the skin with traumatic neuritis of the superficial nerves and secondary vasodilatation. It occasionally will result from injury to the ligaments, discs, bones, and nerves of the neck or from adhesions involving pain-sensitive structures in the arachnoidea. However, the great majority resemble the type of headache resulting from the stresses of life situations and unrelated to head trauma.[2] "Compensation neurosis" and malingering may be important in some cases.

*Post lumbar puncture headache.* Lumbar puncture headache in the erect position results if 20 ml or more of cerebrospinal fluid is

removed.[2] The headache diminishes in intensity or disappears in the reclining position, if the head is held in flexion or extension, or if the spinal fluid is replaced with an equal amount of sterile saline. Although less fluid may be removed during the usual diagnostic lumbar puncture, considerably more may escape from oozing through the hole left by the puncture needle. With loss of fluid, there is an alteration in the hydrodynamic equilibrium, and vessels, especially veins, become dilated. With a portion of its fluid cushion removed, the brain sags slightly downward and traction is exerted on pain-sensitive structures. Such headaches usually disappear within 7 to 10 days. If the recumbent position is assumed, relief is invariably produced. Analgesics are not usually successful.

*Postpneumoencephalography and postventriculography headache.* Withdrawal of spinal fluid and replacing it with air results in distortion of pain-sensitive structures and headache. The treatment is the same as for post lumbar puncture headache. Sedation may be of benefit.

*Dehydration headache.* Severe dehydration may cause headache as the result of low cerebrospinal fluid volume, sagging of the brain, and traction on pain-sensitive structures.[23] It is relieved by rehydration.

## SUMMARY

The problem of headache has been discussed. A classification, together with the clinical characteristics, mechanisms, etiology, and treatment of headaches, is presented.

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## THE USE OF NEW DRUGS

The thoughtful physician will use drugs with the full realization that in doing so he is taking a calculated risk, weighing the chances for cure or relief against the ever-present possibility that harm may be done. . . . Caution is especially necessary in the use of newer drugs. . . . Scientists inevitably accumulate facts faster than understanding; one of our obligations is to realize this and exert our individual and collective efforts toward advancing understanding.—JESSE D. RISING: Medicines, Miracles and Mischief. *Postgraduate Medicine*, September 1958.

# Indirect Inguinal Hernia and the Internal Ring

LIEUTENANT COLONEL STROTHER B. MARSHALL, USAF, MC

FEW PROCEDURES have occupied the thought and effort of surgeons as has the repair of inguinal hernia. Yet the recent review by Koontz,[1] who polled members of the American Surgical Association and the Southern Surgical Association on basic aspects of hernia surgery, indicates a wide divergence of opinion. Careful dissection and high ligation of the hernial sac have traditionally been recognized as the sine qua non of indirect hernia repair. Subsequent steps in the repair, however, have been treated in so many different ways that eponymic procedures have drawn emphasis away from the varying-sized but omnipresent defect in the transversalis fascia—from what Koontz believed is "the core of the whole matter . . . that great care should be used in closing the internal ring."[1]

## REVIEW OF THE LITERATURE

The fact that "there is . . . no unanimity of opinion as to the best type of operation to be used for the ordinary, run-of-the-mine, primary indirect inguinal hernia"[2] is perhaps best explained in part on a historic basis. The importance of the transversalis fascia as the major defensive barrier for the abdominal wall in the inguinal region has long been known. Many years ago, Cooper[3] described this layer clearly in his dissections. Halsted[4] was careful to include it as the deep layer in his original through-and-through suture, which incorporated both oblique muscles and the transversus abdominis as well. Bassini[5] used it in his triple layer suture, incorporating internal oblique, transversus, and transversalis fascia. However, from the time of Bassini, the adoption of a suture line between the internal oblique

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muscle and Poupart's ligament initiated a tradition from which hernia surgery has not yet fully recovered.

Along the road, a group of purists, anxious to repair indirect inguinal hernia by returning the inguinal canal to its normal state, followed the path of Marcy,[6] who in 1871 first sutured together the transversalis fascial margins of the defect at the internal ring beneath the cord. In 1909, Connell [7] stressed the importance of closing the internal ring as a separate layer. By 1928 he had found 9 other authors presenting arguments for this approach.[8] Foremost among these were Andrews [9] and Pitzman,[10] both of whom presented the case for closure of the internal ring in the clearest prose and with profound knowledge of the anatomy involved. During the past 30 years, this concept has had staunch advocates.[11-14]

Several surgeons [15-16] have been satisfied with closing the ring by suturing the medial margin of the transversalis defect to the inguinal ligament laterally, thereby leaving a defect at the superolateral aspect of the cord as it emerges through the abdominal wall. Attention has recently been drawn to recurrences at this site.[17] In virtually all indirect hernias, however, there is sufficient tissue making up the lateral crus of the transversalis fascia to effect a closure using this layer alone without recourse to the inguinal ligament. By retracting the inguinal ligament and using the femoral sheath as the lateral margin, McVay and Chapp [11] have had excellent results in a large series of cases of small indirect hernia. It is less important that the lateral suture include fibers of the inguinal ligament than that it be of sufficient depth to include the lateral crus, thus truly closing the ring.

Subsequent to suture of the transversalis fascia beneath the cord, the role of the overlying internal oblique muscle in strengthening the repair has received attention from all proponents of internal ring closure, from the days of Marcy [6] and Ferguson.[18] Impressed by this action of the muscle, MacGregor [19] used the term "internal sphincter" and devised a technic of suture through the transversalis fascia, with a recurrence rate of 1.8 percent in 111 patients followed.[20] There is little doubt that placing the ring as high as possible beneath the internal oblique through elongation of the inguinal floor helps considerably to protect this vulnerable area in the inguinal canal. During contraction, the internal oblique further occludes the opening and its overlying muscle bundles become a real barrier to intra-abdominal pressure.

The importance of closure of the internal ring is more striking when one considers recurrences following hernia repair. In 1951, Levy, Wren and Friedman [21] reviewed 1,282 cases of recurrent hernia reported in the surgical literature up to that time and found that an

average of 52.3 percent had recurred as indirect hernias. In a similar study, Zawacki and Thieme [17] reviewed 105 recurrent hernias for site and type of recurrence. Again, 52 percent had recurred at the internal ring. Watson,[22] also, has stated that "after the Bassini operation, indirect inguinal hernias most frequently recur through the opening left for the cord." Levy and his colleagues [21] summarized a long-felt need in concluding that repair of all inguinal hernias should include "careful search for an indirect sac; high ligation of the sac, when found; and snug closure of the internal ring about the cord, using the deepest tissues available for repair."

Extending the classification of Ogilvie,[14] Harkins [23] has recently presented a logical and orderly approach to the consideration and teaching of hernia surgery, outlining four types of repair, each successive step incorporating the previous ones. Type 1 consists of simple ligation of the sac; type 2 adds suture of the transversalis fascia to give a snug closure of the internal ring; type 3 adds imbrication of the transversalis fascia over the direct space; and type 4 adds suture of the conjoined tendon to Cooper's ligament. For a number of years McVay and Chapp [11] have utilized only two basic operations: abdominal ring closure for small indirect hernia and Cooper's ligament repair for all others.

**Hernias in Childhood**

The question of how much surgery is necessary in hernia repair in children has recently received much attention.[24-27] In pediatric centers a great deal of emphasis has been placed on simple ligation of the sac without disturbing the cord and, consequently, without demonstrating or disturbing the internal ring. The quoted recurrence rates have been extremely low, in spite of the fact that those who have inspected the rings in infants have commented that at operation they are at times widely dilated by the pressure of a large sac. It has been postulated that subsequently the transversalis fascia tightens with the growth of the child much as small umbilical defects close spontaneously with growth. At what age this process stops and whether it takes place at all have yet to be demonstrated. Koontz [25] has stated, however, that "in addition to ligating and excising the sac, the little hole in the abdominal wall ought to be closed." Potts, Riker and Lewis,[24] who reported a single recurrence in 200 repairs followed for several years, stated that no repair was used at the internal ring unless the sac was "enormous" or "admits two fingers." It would seem that the goal of inspecting the internal ring by dividing the cremasteric muscle attachment, even in children, would assure a high

ligation of the sac—that is, above the internal ring. Furthermore, as emphasized recently by McVay and Chapp,[11] the one or two sutures necessary to close the ring would add little in time or complication for the insurance it would offer. A recurrence in a child, even at a much later date, might thus be prevented.

## REVIEW OF CASES

In the present report, 88 consecutive operations for indirect hernias performed from August 1957 to February 1959 are reviewed. This includes 47 children and 41 adults (table 1). Recurrent hernias were included if they presented at the internal ring, thus appearing as indirect hernias. In all cases the sac was ligated above the internal ring and the cord elevated from the transversalis fascia floor at the internal ring. This necessitated dividing most of the cremaster muscle

**Table 1.** ***Type of operation and distribution in 88 cases of indirect inguinal hernia, August 1957 to February 1959***

| Type of operation | Age | | |
|---|---|---|---|
| | <1 yr | 1–16 yr | 17–44 yr |
| Ligation of sac | 1 | 3 | 0 |
| Ligation of sac and closure of ring | 19 | 22 | 3 |
| Ligation of sac, closure of ring, and imbrication of transversalis fascia | 0 | 2 | 38 |
| Total | 20 | 27 | 41 |

at this level, reducing the cord to its basic elements of vas and vessels. After ligation and division of the sac, the peritoneal stump was allowed to retract freely. The cord was elevated and retracted medially and superiorly. The internal ring was then inspected for size. Whenever it was necessary to create a snug closure, the ring was closed beneath the cord by suturing the transversalis fascia only with interrupted sutures of nonabsorbable material (fig. 1). In adults, in addition, the direct space was evaluated with a finger within the peritoneal sac; and when indicated, imbrication sutures were placed in the transversalis fascia from the pubic tubercle to the internal

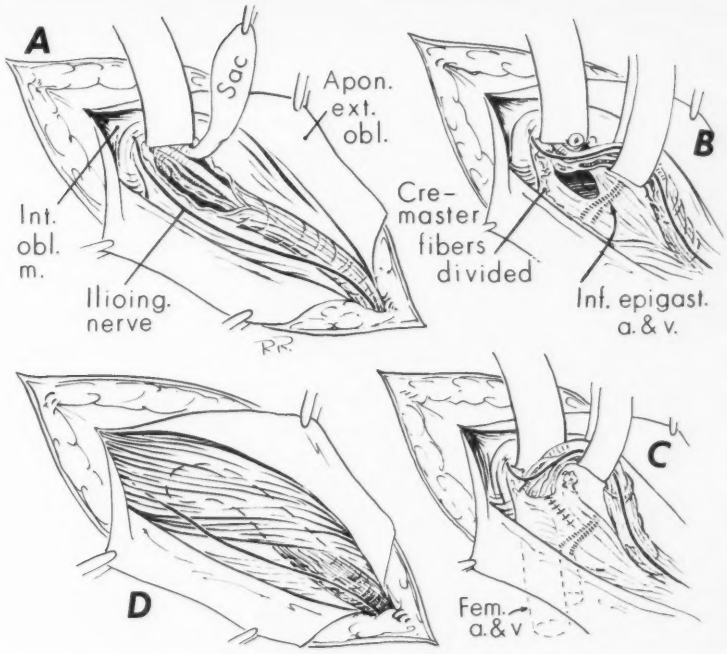


**Figure 1. Successive steps in closure of internal ring. (A) Longitudinal separation of the cremaster muscle and liberation of the sac. (B) Ligation and excision of the sac. Cremaster fibers are divided only at the level of the internal ring, to ensure clear visualization of the fascial margins. (C) Closure of the ring. Fine interrupted sutures of silk or cotton are used. On completion, no more than the tip of a fine hemostat can be passed through. (D) Final location of the internal ring beneath the overlying internal oblique muscle.**

ring, strengthening and reinforcing the original closure of the internal ring at this level.

### Discussion of Recurrences

To date, 82 of the 88 cases have been followed. The only recurrence was in a 5-year-old boy, operated upon on 31 March 1958 for right-sided hernia of 1 month's duration. At the time of operation, the hernial sac was of small diameter and the internal ring appeared to measure less than 1 cm. This was one of the 4 patients on whom no repair of the internal ring was performed. Recurrence was first

noted 14 months later, and repair was performed on 18 June 1959. After ligation of the new sac, the internal ring was found larger than suspected, requiring 4 sutures for closure beneath the cord.

Thus, in those cases treated by closure of the internal ring, there have been no early recurrences. The significance of this is admittedly limited, since it is well recognized that a period of 10 to 20 years is necessary for determination of a true recurrence rate. Most surgeons, however, have found that about 50 percent of recurrences appear within the first year of operation and 25 percent within 6 months.

Of the 47 children in this report, 3 presented as recurrent indirect hernias. A summary of these follows:

## CASE REPORTS

CASE 1, MM #68 978. A 4-year-old boy had undergone repair of bilateral inguinal hernia at 1 year of age at another hospital. The hernias on both sides had recurred by age 2, at which time right herniorrhaphy was performed. The left side was repaired by us on 3 September 1958. A large sac with a single silk suture was identified about 2 cm from the internal ring. After high ligation and excision of the sac, a very large defect at the internal ring was repaired.

CASE 2, AA #70 606. A 14-year-old boy had undergone operation at 4 years of age for a right inguinal hernia. Recurrence with incarceration 7 months prior to admission had required reduction by a physician. Repair was performed on 5 January 1959. A small sac was identified on the superomedial aspect of the cord. Four sutures were necessary to close the internal ring.

CASE 3, SF #69 113. A 21-month-old boy had undergone repair of an inguinal hernia on the right at 5 weeks of age at another hospital. Following an illness characterized by much coughing and vomiting, recurrence was noted 2 weeks prior to admission. Repair was effected on 13 September 1958. A moderate-sized sac with a wide base was found. There were no adhesions in the region of the internal ring. After ligation and excision of the sac, three sutures were required to produce a snug closure.

## SUMMARY

The methods of handling indirect inguinal hernias are discussed with emphasis on the necessity for inspecting and closing the internal ring after high ligation of the sac. A series of 88 consecutive operations for indirect hernia is reviewed. Among the 82 patients who have been followed for 18 months, the only recurrence has been in a child, one of 4 patients in whom the internal ring was not closed.

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# Hysterical Abdominal Bloating Not Due to Gas

FRANK J. LEPREAU, JR., M.D.
LIEUTENANT ALFRED W. WOLFSOHN, MC, USNR

WE CALL ATTENTION to an old clinical entity which is not commonly recognized and report three illustrative cases. This is an hysterical type of abdominal bloating without gas, due to involuntary protuberance of the abdominal muscles, arching of the back, and perhaps flattening of the diaphragm. A careful history will usually elicit an emotional basis for these manifestations. Competent, sympathetic, and unhurried discussion with the patient and her family will often ameliorate the symptoms.

In 1881, Mitchell [1] reported the case of a woman who bloated and knew the swelling had nothing to do with gas, but was caused by emotion. Alvarez [2-4] vividly reported his personal observations in 92 cases and summarized the literature. There have been no important contributions since his last article.

Most of these patients are severely neurotic women, 30 to 50 years of age. They have often had multiple abdominal operations. The swelling will come and go suddenly: "I go up and down like an accordion," one patient stated.[3] The attacks become increasingly frequent and long lasting. Clothes no longer fit. The women may believe themselves pregnant, but the normal menstrual cycle belies this. Pseudocyesis is one form of the entity. There is no passage of gas by mouth or rectum to account for the sudden deflation. Pain may be absent, but more likely there will be some type of discomfort or misery, such as a feeling of burning or bursting.

Physical examination shows a healthy woman, sometimes overweight. The distention is tense or soft. Ascites is often suspected. The swelling may be asymmetrical. Peristalsis is normal. All roentgenographic examinations are normal. While the patient is in the

From Truesdale Hospital, Fall River, Mass., and U.S. Naval Hospital, Newport, R.I. Dr. Wolfsohn is now at the Yale-New Haven Medical Center, New Haven, Conn.

hospital the quickest way to make the correct diagnosis is to administer an anesthetic and note if the abdominal girth is suddenly lessened. Soon after his chloroform discovery, Simpson [5] used this test for spurious pregnancy. "In one very marked case in the hospital, he (Simpson) had passed a tube per anum, its nozzle being kept under water, but not a bubble of air escaped." In 1958, we used thiopental sodium, omitting the rectal tube but with all bystanders attentively listening and watching.

## CASE REPORTS

CASE 1. A 37-year-old woman, complaining of intermittent abdominal swelling, was referred to the Truesdale Hospital by Dr. William Serbst.

Three and one-half years before she had sudden enlargement of the abdomen, which subsided spontaneously after 2 or 3 days without medication. This condition recurred frequently until January 1958, when the episodes occurred twice in 1 month. By 15 April 1958, the condition had become almost constant and was accompanied by epigastric pain and nausea. The patient's appetite was good. There was no vomiting. Bowel movements were normal. There were no urinary symptoms, no loss of weight, and menstrual periods had always been normal.

Physical examination showed a well-developed, well-nourished, and healthy-appearing woman. The positive findings were limited to the abdomen, which was tense without shifting dullness or masses and with normal peristalsis. There was minimal epigastric tenderness. Rectal examination findings were normal. Vaginal examination revealed a relaxed introitus, a normal cervix, and a small anteverted uterus. All roentgenograms were normal. These examinations included abdominal films, erect and supine when the patient was distended, cholecystograms, gastrointestinal series, barium enema, intravenous pyelograms, and roentgenograms of the chest. All laboratory studies including examination for urinary porphyrins were normal.

The following consultation note was written by Dr. Everett Radovsky*:

"The history reveals an interesting sequence of events which appear to be causally related, beginning with a tubal ligation performed 7 years ago. This was urged on the patient because of recurrent phlebitis with two previous pregnancies. She reluctantly consented in spite of her religious beliefs because of close rapport with her physician. For the week prior to surgery, she cried constantly. Afterward, she had strong feelings of guilt and of having committed a sin. Although she was a staunch Catholic, she was afraid to go to confession. Her family finally prevailed upon her and 5 years ago, 2 years after the operation, she went to confession. Even though she had confessed, and repented sincerely and persistently, she has been unable to give up these guilt feelings.

"Since the operation, she has suffered from a state of frigidity, having neither satisfaction from nor interest in sexual relations. This seems to be related to other facets of the problem in two ways: 1. If she enjoyed sexual

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*Chief, division of general practice, Truesdale Hospital, Fall River, Mass.

relations she would be having intercourse without being subject to the primary purpose of it, conception. 2. At the same time, by not willingly entering into sexual relations she is depriving her husband, 'who is better than she deserves,' of his marital rights.

"It is my impression that the patient is a deeply religious person in whom a tubal ligation precipitated feelings of guilt because of her sinfulness in the eyes of her church and her inability to meet her marital obligations. I believe that subconsciously she has the feeling that if she could become pregnant, this would remove the need for her to feel sinful or to withhold herself from her husband. This would place her situation within the scope of pseudocyesis. She has heard of people who become pregnant following a tubal ligation, but unfortunately not this long after. I believe that if the patient can be reassured that she has sufficiently expiated her sin, she may be able to overcome her guilt feelings and the need for her abdominal symptoms would no longer exist. Diagnosis: Anxiety hysteria. Recommendations: Insight and reassurance."

As a diagnostic test, thiopental sodium was administered on a morning when the patient's abdomen was moderately distended. The abdominal circumference at the umbilicus was 35 inches. Atropine, 0.4 mg, and Demerol hydrochloride, 100 mg, were administered. After one-half hour there was no change. Flexion of the thighs made no difference. Thiopental sodium was given intravenously and immediately the abdomen became flat. The lumbar lordosis disappeared. The circumference was now 33 inches at the umbilicus.

The patient was discharged from the hospital after 12 days of study with the recommendation that she and her family physician arrange for consultation with the proper religious advisor.

The patient at first did well, having gained some insight into her problems. During another bout of swelling, however, she consulted a different surgeon. An exploratory laparotomy was negative and revealed nothing to explain the recurrent distension of her abdomen.

CASE 2. A 30-year-old unmarried sailor was admitted to the U.S. Naval Hospital, Newport, Rhode Island, for the second time in February 1958 because of abdominal bloating which had occurred intermittently for four years. He stated that the distention had been much worse for the past 2 years after he was disciplined, considered a liberty risk, and not allowed ashore in many ports. The bloating would last for 2 or 3 weeks. After it set in, he actually lost weight as a result of mild anorexia. There was no bowel dysfunction. When the patient was relaxed, the bloating suddenly left him and he passed no flatus. The episodes of distention were associated with back pain. Although the patient has been a moderately heavy drinker of alcohol for the past 10 years, the latter did not relieve his bloating. He became very irritable and pugnacious. His prior admission to the hospital was because of abdominal distention and a history of several bouts of dark urine and light stools over a 3-year-period. Physical examination and laboratory studies, including a liver profile, urine for bile and urobilinogen were negative at that time. A liver biopsy revealed fatty metamorphosis. His history of dark urine and light stools was never objectively substantiated clinically or in the laboratory on any hospital admission.

Physical examination revealed an obese white male in no acute distress. Vital signs were normal. There was neither scleral nor general jaundice. The liver was palpable two fingers below the right costal margin. It was thought that the patient had a fluid wave.

The laboratory studies revealed a normal blood cell count and urinalysis. The urine was negative for bile and urobilinogen. Six urine specimens were negative for porphyrins and porphobilinogen. Electrolytes were normal. All liver function studies as well as basal metabolism rate, protein-bound iodine, fasting blood sugar, and heterophil antibody test were negative. A roentgenogram of the chest revealed normal findings, and a flat plate of the abdomen was negative and showed no abnormal amount of gas when the patient was distended. Upper and lower gastrointestinal series were normal.

Multiple abdominal paracenteses yielded no fluid. Sigmoidoscopy was negative. Exploratory laparotomy revealed a large amount of fat subcutaneously and in the omentum. A normal Meckel's diverticulum and appendix were removed. An open biopsy of the liver revealed fatty metamorphosis. Postoperatively, during an episode of distention, the patient had a wound disruption, which was repaired, and he made an uneventful recovery.

Inasmuch as the patient continued to complain of distention, he was given 300 mg of thiopental sodium intravenously. Complete relaxation of his abdomen resulted, the circumference of his abdomen diminished by two inches, and his lordosis disappeared. The same effect, but to a lesser degree, resulted after the injection of mercurials and placebos.

With superficial psychotherapy, the abdominal distention has markedly diminished, and the patient has returned to active duty.

CASE 3. A 41-year-old woman had been seen for numerous complaints, including abdominal, back, and chest pains in the outpatient clinic of the U.S. Naval Hospital, Newport, Rhode Island. On two occasions she presented as an acute anxiety state. One year previous, dilation and curettage of the uterus had been normal. For the past 1½ years she had had intermittent bloating of the abdomen, lasting a few days every month. At times this bloating was related to menstrual periods but more commonly not. There was no passage of flatus when the abdomen deflated. She had no food intolerance. She often vomited on arising. She had been successfully treated for syphilis. The patient had a 23-year-old daughter by her first husband, who was killed at Pearl Harbor. She remarried in 1952 and had tried to have another child. She adopted one of her daughter's three children 5 years ago. The bloating of the past 2 years had coincided with a desperate attempt to become pregnant.

The only positive finding on physical examination was a distended abdomen. A complete blood cell count, sedimentation rate, urinalysis, and Kahn test were negative. Roentgenograms of the chest, upper and lower gastrointestinal series, and gallbladder series were all normal. The abdominal flat plate when the patient's abdomen was distended revealed no abnormal gaseous pattern. The patient was thought to have hysterical abdominal distention without gas. Her bloating has become much milder and less frequent as a result of greater insight into her problem.

## SUMMARY

Hysterical abdominal bloating not due to gas is the result of an involuntary protuberance of the abdominal muscles and arching of the back. A careful history will usually elicit an emotional basis. Most of the patients are women between the ages of 30 and 50 years.



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They often have had multiple abdominal operations. In these patients there is a normal menstrual cycle. There is no passage of gas by mouth or rectum to account for the sudden deflation that can be brought about by heavy sedation or anesthesia. Three cases of hysterical abdominal bloating without gas have been presented in the hope that the cause of the condition will be recognized more frequently. The diagnoses can be made if one is aware of this entity and organic pathology is excluded.

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## OBSOLETE VERSION OF BEDSIDE MANNER

After Harvey's discovery of the circulation of the blood in the early seventeenth century, medicine passed through a period of great flux, and progress was influenced both by philosophy and by the new science. It was in the eighteenth century that the gold-headed cane became the physician's symbol of office and helped to impress the patient. This was important because there was considerable cynicism about the doctor's capabilities, and indeed Voltaire believed that the physician was a man who poured medicine of which he knew nothing into a body of which he knew less. Nevertheless, at that time the London physician was often rich and his social standing high, but in contrast the rural practitioner was humble and of modest means. Content to ride his horse over miles of bog and moor every year for perhaps £200, he had to be physically strong and mentally alert. Many of his patients were desperately poor, and it is said that Dr. Fothergill, the Quaker physician of that time, often made the act of feeling the pulse a cover for slipping money into the patient's hand.—JOHN LISTER: United We Stand—Divided We Fall. *New England Journal of Medicine*, March 26, 1959.

# Clinicopathologic Conference

**U.S. Naval Hospital**
**Philadelphia, Pennsylvania**

A 63-YEAR-OLD WHITE MALE VETERAN was admitted to this hospital on 25 July 1957 with symptoms of weakness of the right side, confusion, and inability to speak. The onset of these difficulties had occurred suddenly on 14 July 1957, and he had been treated at another medical facility for approximately 1 week and sent home, improved. Admission to this activity 3 days later had been prompted by a recurrence of symptoms. During the 24 hours prior to admission he complained of severe headache with repeated forceful vomiting and became irritable, restless, and subsequently drowsy. The vomitus was described as dark brown, apparently free of blood. He also developed continuous hiccup.

The past history, obtained from the patient's wife, revealed that he had retired as a bricklayer and that during the past several years he had worked only at gardening as a hobby. He had enjoyed good health except for chronic complaint of abdominal pains and biparietal headache during the past 8 years. The abdominal pain was present morning and evening and relieved by bowel movement. The patient's wife could not furnish more specific information in regard to the headaches. Details of the family history were considered not significant.

## Physical Examination

The patient was an elderly, well-nourished, white man who did not appear chronically ill. The blood pressure was 140/80 mm Hg; pulse 64 per minute and regular; respiratory rate 16 per minute. He had continuous hiccup. Auscultation of the precordium revealed a midsystolic click, maximal at the pulmonic area, which disappeared on full inspiration. A relaxed left inguinal ring was evident without any hernial mass. Neurologic examination revealed a mental state

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From the laboratory service, U.S. Naval Hospital, Philadelphia, Pa., Capt. Bruce H. Smith, Jr., MC, USN, chief. Capt. Charles L. Ferguson, MC, USN, former commanding officer, was succeeded by Capt. Edward T. Knowles, MC, USN, on 10 September 1959.

characterized by drowsiness, with restlessness and irritability when disturbed. He was aphasic, with garbled speech. Slight nuchal rigidity was present. The optic fundi were normal. A right hemiparesis, including a central facial palsy and involving the upper and lower extremities about equally, was present. Right hemihypesthesia was noted. Gross confrontation studies of the visual fields suggested a right homonymous defect. Because of the patient's mental state and aphasia, no attempt was made to substantiate this latter finding by perimetry and tangent field studies. The deep tendon reflexes were equally hyperactive in the right upper and lower extremities and an equivocal extensor plantar reflex was elicited on that side. The abdominal and cremasteric reflexes were absent on the right.

**Laboratory Studies**

Routine laboratory studies including complete blood cell count, urinalysis, blood sugar, blood urea nitrogen, and Venereal Disease Research Laboratory (VDRL) serologic test were negative or within normal limits. After funduscopic examination revealed no evidence of papilledema, lumbar puncture indicated an initial pressure of 330 mm of water. Examination of the spinal fluid showed 2 cells (lymphocytes) per cu mm, sugar 63.4 mg per 100 ml, chloride 117.5 mEq per l, protein 55 grams per 100 ml.

**Course in Hospital**

It became evident with observation of the patient's clinical course that the altered mental state and neurologic deficits were characterized by progression. Roentgenograms of the chest (fig. 1) disclosed a minimal amount of strandlike increased density in the right base, just above the right hemidiaphragm. There was a slight shift of the heart and mediastinum to the right. A definite conclusion could not be reached inasmuch as these findings could represent either an inflammatory process or an underlying bronchogenic malignancy. Examination of planigrams (fig. 2) revealed a constricting lesion in the distal portion of the right lower lobe bronchus below the level of exit of the middle lobe bronchus. There was a soft tissue density, which appeared to protrude into the bronchus below the exit of the upper lobe bronchus. These findings were interpreted as highly suggestive of a malignancy in this area. Skull roentgenograms were normal. A left carotid arteriogram (figs. 3 and 4) showed good filling of the anterior cerebral and middle cerebral vessels. The anterior cerebral artery was displaced to the right in its anterior portion, believed suggestive of a frontoparietal mass.

On the ninth hospital day a left craniotomy was performed. Following exploration by means of repeated passes with a ventricular needle through the posterior frontal, temporal, and parietal regions, a biopsy specimen was obtained from deep within the posterior frontal area, where increased resistance within the white matter had been encountered. Microscopic examination of this biopsy tissue revealed gliosis and satellitosis.

Within the immediate postoperative period and for several weeks following, an improvement was observed in the patient's mental alertness, but no significant improvement in the aphasia or hemiparesis could be discerned. During this period a subtemporal decompression performed at the time of surgery remained soft. The terminal few weeks of hospitalization were characterized by progressive mental

**Figure 1. Posteroanterior roentgenogram of the chest, showing elevation of the right hemidiaphragm and increased density in the right base.**

**Figure 2. Planigram of chest, showing constricting lesion of right lower lobe bronchus and soft tissue density protruding into lumen.**

deterioration with evidence of increased intracranial pressure. The patient died 3 months following onset of symptoms.

## DISCUSSION

DR. WHITMORE:* The site of the lesion or lesions seems obvious and it appears that the more accurate delineation of their true nature would pose the main problem in this case.

The history is that of a 63-year-old white man whose original presenting signs and symptoms were apparently attributed to a fairly

*Lt. John T. Whitmore, MC, USN, resident, medical service.

**Figure 3. Arteriogram, showing shift of left anterior cerebral vessels to the right.**

typical cerebrovascular accident involving the left middle cerebral artery. Approximately 2 weeks later he was beset with a recurrence of these signs and symptoms, precipitating his admission to this hospital. Noteworthy details of his history as obtained on this admission included severe headache, forceful vomiting, and continuous hiccup. The physical findings were compatible with an intracranial lesion involving the dominant left cerebral hemisphere. The cerebrospinal fluid pressure and the findings on the left carotid arteriograms prompted craniotomy with the hope of finding a surgically benign intracranial lesion. The findings at craniotomy led to the assumption that the lesion was inoperable.

Posteroanterior chest film and planigrams were interpreted as revealing a constricting lesion in the distal portion of the right main stem bronchus and were considered highly suggestive of malignancy in this area. I assume that no attempt was made to perform bronchos-

copy on this patient for a biopsy diagnosis because of his poor general status. It is unfortunate that this could not have been done, for a tissue diagnosis of bronchogenic carcinoma of the lung would provide a reasonable primary site. Cancer of the lung produces cerebral metastases in a remarkably high percentage of cases, the reported incidence ranging from 20 to 40 percent.[1] Most disconcerting clinically are those not uncommon cases in which the chief complaint and presenting symptoms are due to the cerebral metastases. These metastases are carried via the arterial blood stream as small tumor emboli, which may or may not—depending upon their size at the time of migration—cause vascular symptoms. The symptoms may vary from a psychosis to a hemiparesis of sudden onset and are unfortunately not peculiar to metastatic lesions as distinguished from primary tumors or from vascular disease of the brain. The mode of metastasis, the usual age group of these patients, and the well-documented statistical advantages of routine chest roentgenograms indicate the importance of considering cerebral metastases in the differential diagnosis of all apparent cerebrovascular accidents as well as in the

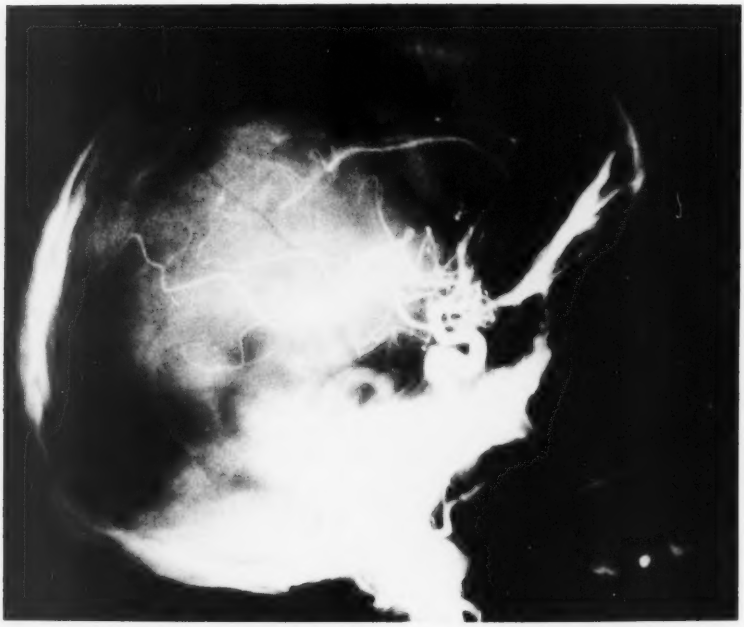

**Figure 4. Arteriogram, lateral view.**

preoperative evaluation of "cerebral tumors." The possibility of a metastatic brain lesion from a quiescent primary, especially one arising from the lung, breast, or kidney, must always be considered. Craniotomy in this instance, I presume, was performed to exclude a second and entirely unrelated intracranial lesion.

Nevertheless, in the absence of a proved bronchogenic lesion, due consideration must be given to a differential diagnosis of intracranial mass lesions. I would classify these according to a vascular, traumatic, infectious, or neoplastic etiology.

The normal skull roentgenogram and normal cerebrospinal fluid findings with the exception of increased intracranial pressure would mitigate to various degrees against the presence of either thrombosis, hemorrhage, or embolism. On the other hand, there are those few instances of intracranial vascular lesions whose presence offers no clue in the cerobrospinal fluid. I believe, however, that compared with other possible etiologic agents, the evidence for any vascular lesion, except subdural hematoma or intracerebral hemorrhage with clot formation, is insufficient. The history, physical findings, and laboratory results are compatible with the presence of either of these latter 2 lesions. I would delete intracerebral hemorrhage from consideration in this instance not because of incompatibility, but because of the operative findings and the lack of the usual cerebrospinal fluid findings.

There was no history of head injury and no evidence of trauma on physical examination or skull x-rays; there is therefore no reason to implicate a traumatic lesion other than subdural hematoma. However, it would seem that at craniotomy subdural hematoma was exonerated as a cause. The vagaries of subdural hematoma as regards position and the correlation of signs and symptoms might still allow for some suspicion of its presence, but I do not believe that one could expect more evidence against its presence than the findings at craniotomy in this case.

In regard to infection as the cause of hemiparesis, intracranial infectious lesions are most frequently the result of thrombosis of inflamed vessels and are more common in chronic and subacute infections such as tuberculosis and syphilis.[2] Normal cerebrospinal fluid studies, the absence of a history indicting these chronic infections, a negative blood serologic examination, and the absence of a febrile course would all lend little support to an etiologic agent from this group.

Tumors, neoplastic primary or secondary metastatic lesions, and brain abscess would seem plausible causes of increased intracranial pressure, recurrent focal neurologic findings, and the abnormal left carotid arteriogram in this patient. The classic signs of increased intracranial pressure, nausea, vomiting, and severe headache were

present, and were corroborated by the cerebrospinal fluid pressure of 330 mm of water. In addition, attention may be directed to the presence of continuous hiccup, which in this particular case could be postulated as due to intrathoracic or intracranial involvement or irritation. Tumors in the vicinity of the fourth ventricle have been known to cause hiccup and might also account for the presence of nausea and vomiting on the basis of irritation of the vagus nerve. The absence of papilledema, the most characteristic single sign of brain tumor, does not rule this diagnosis out; and papilledema may be absent in approximately one quarter to one half of cases.[2] The findings on arteriography and at operation would seem to offer substantial evidence in this patient of an intracranial tumor.

The diagnosis of brain abscess may be entertained whenever there is a focal brain lesion and a logical source of sepsis, usually pulmonary or cranial. An afebrile course, as implied in this instance, would remain consonant with a diagnosis of brain abscess in that fever is not usually present unless the focus is active. In addition, whether the pulmonary findings represent malignancy or not, it could be surmised that there was some degree of pulmonary infection superimposed upon a malignancy at one time or another, thereby providing a source of sepsis for infective embolization.

This approach narrows the differential to include metastatic tumor to brain, primary brain tumor, and brain abscess. In the presence of the pulmonary findings as described, I feel the most logical diagnosis in this age group would be a primary bronchogenic carcinoma with metastases to the brain.

Before closing I would also comment on the complaint of abdominal pain, which seems unrelated and most readily accounted for on the basis of a greatly relaxed left inguinal ring. Brain tumor may be associated with abdominal pain, the mechanism or causal relationship for which I do not know and have not found described.

DR. BRANNON:* From a clinical standpoint we were presented with an elderly person whose history had related a sudden onset of speech deficit, nausea, vomiting, and a weakness of the right side. From that time there had evidently been a progression of symptoms, with weakness of the right side a prominent feature. This man had retired some years ago and had confined himself almost exclusively to gardening. On one occasion, following discharge from his initial hospitalization and a few days prior to readmission, the wife had noted that he continued to work in the garden on his hands and knees; yet she claimed he was unable to stand at the time because of the weakness

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in the right leg. This was considered important inasmuch as the neurologic deficit was not inconsistent with a cerebrovascular accident, as suggested by the history.

We entertained a clinical impression at the time of admission that he had initially suffered a cerebral thrombosis, and that because of his persistent activity after onset of the hemiparesis he had possibly incurred a head injury, with subsequent subdural hemorrhage. A left cerebral arteriogram was performed in the hope of demonstrating such a lesion. As noted previously, this failed to be the case, and a rather marked shift of the left cerebral vascular pattern consistent with an intracerebral lesion was noted. No tumor stain was noted. The pattern of shift in the major vessels was consistent with cerebral edema, which is not uncommonly associated with metastasis. It was also consistent with multiple small metastases. In view of the findings noted on the chest film, a diagnosis of bronchogenic carcinoma with metastasis to the left cerebral hemisphere and associated cerebral edema was considered.

It was decided to turn a generous bone flap to expose the left posterior frontal and temporal lobe, in the hope of revealing an accessible metastatic lesion that could be removed without undue damage to the dominant hemisphere. The exposure failed to reveal such a lesion. A small biopsy specimen was taken from the posterior frontal region, where a suspicion of increased density had been encountered, by passing a ventricular needle through that area. Exploration posterior or deep to this area would have left the patient with a severe neurologic deficit. A moderate-sized subtemporal decompression was resorted to. The patient survived for 2 months following surgery, with total aphasia, progressive mental deterioration to a state of coma, and finally respiratory failure. During this period we continued to consider the primary diagnosis one of bronchogenic carcinoma with metastasis to the brain.

DR. WHITMORE'S DIAGNOSIS:

Bronchogenic carcinoma metastatic to brain

DR. BRANNON'S DIAGNOSIS:

Postoperatively: Metastatic carcinoma, brain, inoperable

## PATHOLOGIC FINDINGS

DR. WOOD:* At autopsy, meticulous search of the bronchial tree failed to reveal the presence of any intrinsic mucosal lesion. There were several enlarged hilar and mediastinal lymph nodes, which in

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the region of the right mainstem bronchus compressed the bronchial lumen at the hilus. The lymph nodes contained large quantities of anthracotic pigment, and there were numerous focal depositions of anthracosilicotic pigment throughout the pulmonary parenchyma. There was no evidence of primary or metastatic neoplasm. There were plaquelike adhesions between the visceral and parietal pleura.

On step section of the brain a primary neoplasm measuring 5.5 cm in diameter was found in the left occipital lobe. The lesion was confined above the tentorium cerebelli and was located in the white matter of the posterior portion of the occipital lobe. It compressed the overlying cortex circumferentially in a symmetrical manner. The adjacent white matter for a distance of 3 cm proximally was cystic and replaced by hemorrhagic brownish tissue. The cortex in these areas was flat and narrowed. The tumor was sharply circumscribed and composed of firm heterogeneous tissue with gross areas of hemorrhage and necrosis. The main tumor tissue was yellow-white in color. The white matter in the involved side was markedly thickened and edematous measuring, 4 cm in the frontal lobe as compared with 1.5 cm at the same site on the right side. This accounts for the clinical finding of shift of the anterior cerebral vessels to the right, which contributed heavily to mislocating the tumor in an area of edema, gliosis, and satellitosis. Microscopically (figs. 5 and 6) the tumor is composed of whorls of pleomorphic cells arranged for the most part around blood vessels. There is associated hemorrhage, necrosis, and endothelial proliferation. Moderate numbers of bizarre mitotic division figures are seen. Special stains show these cells to be fibroblasts of varying stages and for that reason the tumor has been classified as a primary fibrosarcoma of the brain.

Sarcomas of the central nervous system are not common, and few series have been reported. Bennett [3] found 5 sarcomas (1.1 percent) in his series of 446 intracranial neoplasms at the Armed Forces Institute of Pathology. Abbott and Kernohan [4] had 11 intracranial sarcomas available for study. They divided these 11 cases into 3 types: (1) fibrosarcoma (3 cases); (2) perivascular sarcoma (6 cases); and (3) sarcoma of unknown type (2 cases). They believed that these tumors could take origin from any connective tissue within the brain: the adventitia of blood vessels or the pia mater deep in the sulci. Sarcomas of the brain usually are dense and gray, with regions of necrosis. They are invariably sharply circumscribed from the surrounding brain. They seem to be encapsulated because they are commonly surrounded by a zone of necrotic brain tissue, but there is in reality no capsule. Edema of the neighboring brain tissue is common and usually pronounced.[5]

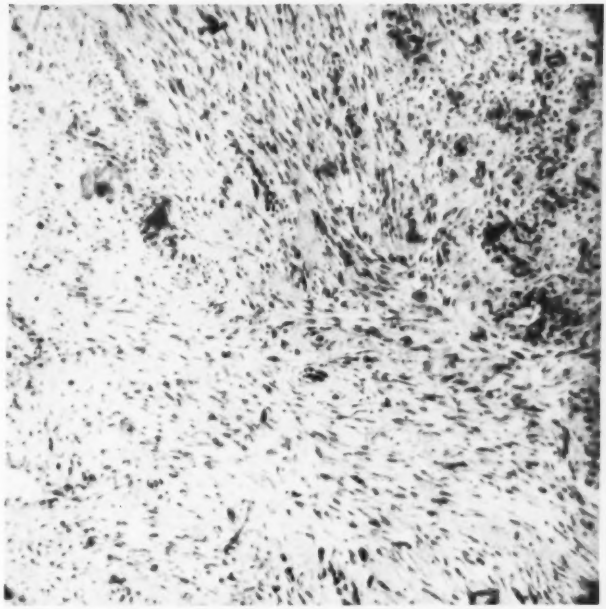

**Figure 5. Representative section of primary brain tumor, showing the whorled configuration and absence of astrocytic cells. (x 42)**

Microscopic examination of the lungs showed moderate congestion and edema, focal lipoid pneumonia, and numerous clumps of dumbbell-like, dark brown bodies with segmented body and central fiber, (fig. 7) considered classic asbestosis bodies. Consultation with the Armed Forces Institute of Pathology [6] confirmed this impression. Similar bodies were found in the hyperplastic hilar lymph nodes.

Asbestosis is a disease resulting from the inhalation of long fibers of asbestos. It is more often acquired by workers in the processing plants than in the asbestos mines, where only the crude asbestos is handled. The fiber consists essentially of magnesium silicate. Because of its relatively large size it does not enter the alveolar sacs but ordinarily lodges in the respiratory bronchioles, where the initial inflammatory reaction occurs. The resulting fibrosis is diffuse, rarely nodular, and involves the basal portions of the lungs rather than the middle portions as in the case of silica. The pleura is involved early and becomes greatly thickened and rigid, usually with obliteration of much of the pleural space by fibrous adhesions. Asbestosis bodies are pathogno-

monic of the disease and are thought to be formed by the deposition of proteins and iron salts on the surfaces of the asbestos fibers. These bodies are segmented fungus-like masses with bulbous ends and are yellow to orange-brown by transmitted light. They are present in the air spaces, surrounded by macrophages and sometimes by multinuclear giant cells, or are imbedded in the dense masses of fibrous tissue, where all alveolar structure is obliterated. Occasionally the bodies can be found in the sputum, making possible a specific diagnosis. Lymph channels are not directly invaded in asbestosis because the particles are generally too large to be phagocytosed and carried in the macrophages to lymph nodes; the involvement of bronchopulmonary nodes is therefore comparatively slight. Obliteration of lymph channels and blood vessels is brought about within involved masses of pulmonary parenchyma and pleura by the same mechanism which operates in any chronic inflammatory process.[7] Since this patient had been a bricklayer and since some kinds of fire brick have asbestos in them, perhaps this was the source of his pulmonary lesion.

**Figure 6. Brain tumor, showing spindle-shaped bipolar cells of fibroblastic nature, with endothelial proliferation and an occasional mitotic figure. (x 130)**

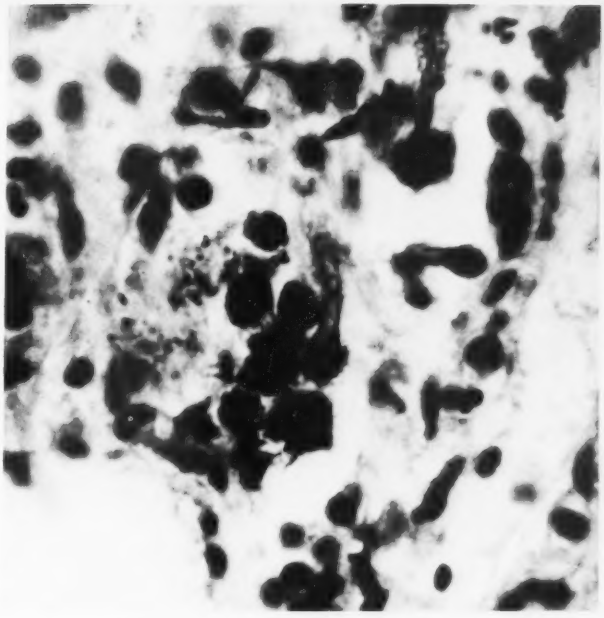

**Figure 7. Characteristic appearance of the asbestosis bodies in the lungs. (x 459)**

PATHOLOGIC DIAGNOSES:

1. Primary fibrosarcoma, left occipital lobe
2. Asbestosis, with involvement of hilar lymph nodes

DR. SMITH: Dr. Brannon, could you now correlate the neurologic and autopsy findings?

DR. BRANNON: One comment Dr. Wood made was in reference to the marked edema associated with this tumor. It is apparent we had miscalculated the location of this tumor both from a clinical evaluation of the patient's neurologic deficit and from interpretation of the arteriogram. The patient's hemiparesis, sensory deficit, and aphasia, as well as the arteriogram pattern, can be ascribed to a space-occupying lesion in the frontoparietal area—in this case edema and cellular reaction rather than the tumor itself. This is a point we have to bear in mind in localizing a neurologic lesion. In a case such as this, with a tumor mass located well posterior in the occipital lobe, the carotid arteriogram failed to reveal evidence to correct the localization.

DR. SMITH: Dr. Mathews, would you comment on the radiographic findings in this case and the usual findings in asbestosis?

DR. MATHEWS:* One of the most striking features of asbestosis is the total absence of radiographic abnormality even when the clinical symptoms are obvious. The patient may be severely dyspneic and asbestosis bodies may easily be found in the patient's sputum, yet his chest radiograph is completely normal.

In asbestosis, the earliest roentgenographic finding is usually the presence of fine strands of fibrosis at the bases. The pulmonary lesions are different from those seen in silicosis, in which pulmonary nodularity is a prominent feature. Conglomerate shadows such as occur with silicosis are usually not found in asbestosis.[8]

The finding of a constricting lesion of the right lower lobe bronchus was more apparent than real, as was proved at autopsy. The associated soft tissue density must have represented a hyperplastic lymph node.

DR. SMITH: Dr. Wood, do you have any final comments?

DR. WOOD: I believe that the gross and microscopic findings of asbestosis with hyperplastic hilar and mediastinal lymph nodes correlate well with the radiographic findings. The elevation of the right hemidiaphragm was due to phrenic nerve paralysis on a central basis from increased intracranial pressure. The cause of death was felt to be respiratory failure due to a combination of increased intracranial pressure, respiratory depression, and a focal lipoid pneumonia, which may have been a terminal event.

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# Case Reports

## Eosinophilic Granuloma of the Rib

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JOSEPH M. MILLER, M.D.
MILTON GINSBURG, M.D.

EOSINOPHILIC GRANULOMA was apparently first described in 1929 by Finzi,[1] with Fraser,[2] in 1935, giving a more complete description of the entity. Lichtenstein and Jaffe[3, 4] named the lesion in 1940. Although the cause is unknown, the disease appears to be closely associated with Letterer-Siwe disease and Hand-Schüller-Christian disease. These three diseases may be different stages of the same condition.[4-10] If this is so, Letterer-Siwe disease, the most acute, generally fatal form, occurs in young children; Hand-Schüller-Christian disease, a severe and often fatal form, in older children; and eosinophilic granuloma, the benign form, in young adults. Biopsies from various areas of the same lesion, however, have shown characteristics of each of the forms of the disease.[7, 8] Fisher[7] reported a change from Letterer-Siwe disease to Hand-Schüller-Christian disease in one patient, and Engelbreth-Holm, Teilum and Christensen[11] observed a change from eosinophilic granuloma to Hand-Schüller-Christian disease in one patient.

Men are affected with eosinophilic granuloma more often than women. The disease usually occurs in children and young adults, although Adams and Kraus[12] reported an instance in a 73-year-old woman.

Involvement of other sites such as the lung,[5, 11-23] kidney,[12] gasserian ganglion,[16] spleen,[24] stomach and small intestine,[25] and skin[26] has been reported. Reports of 15 patients with solitary lesions of the rib[5, 15, 27-37] and 15 patients with involvement of bone and lung[5, 12-23] have been found. One of these patients had an associated spontaneous bilateral pneumothorax.[22]

Serial roentgenograms may show a rapidly growing solitary lesion in a rib. Frequently, a mass may be felt. Occasionally, the lesion is asymptomatic.

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If the lungs are involved, a diffuse, soft, hazy, and occasionally nodular infiltration is seen in the parenchyma during the early stages of the disease. If progression occurs, parenchymal fibrosis appears. Weinstein, Francis, and Sprofkin [23] reported the association of eosinophilic granuloma of multiple bones and infiltration of the lung. Biopsy of tissue from eosinophilic granulomatous lesions of the lung has been performed.[16, 18]

Eosinophilic granuloma may occur as a single lesion or as multiple lesions in bone or soft tissues. Where bony disease is present, the destructive lesions closely resemble neoplastic diseases and inflammatory conditions. A solitary lesion may simulate a bone cyst, a giant cell tumor, a Ewing's tumor, a solitary myeloma, an osteogenic sarcoma, or an osteomyelitis. Multiple lesions may present the same clinical signs and roentgenographic findings as multiple myeloma, metastatic tumor, osteitis fibrosa cystica, Letterer-Siwe disease, or Hand-Schüller-Christian disease.

Multiple diagnostic studies are necessary to establish the nature of a lytic lesion of a rib. A complete blood count is necessary. The values for calcium, phosphorus, acid phosphatase, alkaline phosphatase, total protein, albumin, and globulin of the blood plasma should be determined. The albumin-globulin ratio should be ascertained. A serologic test for syphilis should be done. A urinalysis should be performed and the urine should be examined for Bence Jones protein. Roentgenograms of the chest, skull, vertebrae, ribs, pelvis, humeri, and femurs should be made. A gastrointestinal series, a barium enema, and an intravenous pyelogram may be necessary.

The treatment for a solitary lesion in a rib is excision. Inasmuch as the definite diagnosis usually can be made only after histologic study of tissue obtained from the lesion, a block excision of the overlying muscles, the rib, and the underlying pleura should be performed. The pleural surface of the rib often is eroded, and this additional factor favors such an operation.

Eosinophilic granuloma is sensitive to treatment with roentgen rays.[23] Such therapy should not be given, however, until a piece of the tumor has been obtained for biopsy. Infiltration of the lung has regressed following this type of treatment when given early in the course of the disease.[17, 23] Improvement of parenchymal fibrosis of the lung following such treatment has not been observed roentgenographically.[15, 18, 20]

## CASE REPORTS

CASE 1. A 27-year-old man was admitted to the medical service of this hospital on 19 April 1948 complaining of pain in the left lower part of the back, moderate

shortness of breath of about 4 days' duration, and a nonproductive cough of 1 day's duration. The pain did not radiate.

The patient was well developed but poorly nourished. The physical signs of a left pneumothorax were present.

On 20 April, the hemoglobin was 92 grams per 100 ml. The white blood cell count was 9,100 per cu mm with a differential count of 71 percent neutrophils, 26 percent lymphocytes, 2 percent eosinophils, and 1 percent basophils. On 26 April, the white blood cell count was 7,400 per cu mm of which 70 percent were neutrophils, 22 percent lymphocytes, and 8 percent eosinophils. Serum calcium, phosphorus, acid phosphatase, and alkaline phosphatase were within normal limits. Total serum protein was 7.23 grams (albumin 3.46 grams, globulin

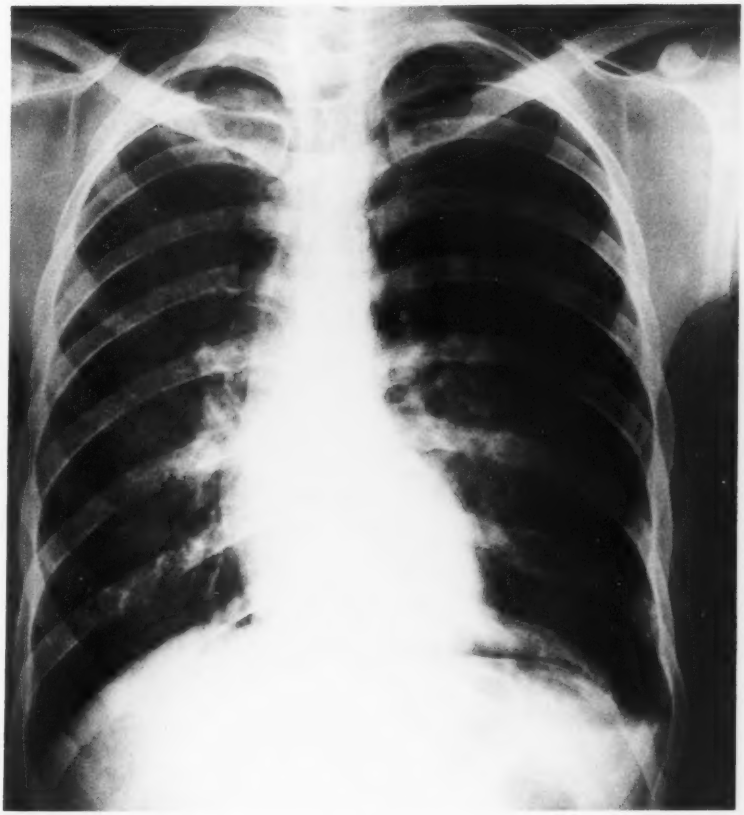

**Figure 1 (case 1). Roentgenogram on 19 April 1948, showing the trachea and the heart displaced slightly to the right side.**

3.77 grams) per 100 ml. The alumin-globulin ratio was 0.91 : 1.00. The Eagle flocculation test for syphilis was negative. The urinalysis was negative. The urine was not examined for Bence Jones protein.

The roentgenogram of the chest on 19 April showed the trachea and the heart to be displaced slightly to the right side (fig. 1). A left pneumothorax with about a 70-percent collapse of the lung was present. A homogenous increase in density was present throughout the collapsed left lung and the right lung had a soft, hazy infiltration in the parenchyma. An area of rarefaction with an expansion of the cortex was present in the posterior portion of the left ninth rib. Roentgenograms of the thorax made by a U.S. Navy facility on 13 and 21 March 1946 did not show the infiltration in the parenchyma or the area of rarefaction in the rib.

The trachea and the heart had returned to their normal position on 29 April. As the left lung re-expanded, a soft, hazy, and perivascular infiltration was seen throughout the lung. The lung was fully expanded on 14 May.

On 17 May the patient was seen by a consultant in surgery, who recommended that the affected portion of the left ninth rib be removed.

About 7 cm of the rib was resected on 20 May. The part contained a fusiform enlargement about 2 cm in width as compared to about 1.3 cm of the normal bone. A soft grayish pink elongated mass, about 1 cm in width and about 2 cm long, was present on the inner surface of the rib. The cortex of the bone was eroded without obvious infiltration of the adjacent tissue. Microscopic examination showed the tumor to be an eosinophilic granuloma.

The roentgenogram of the thorax on 15 June showed an infiltration throughout both lungs with a moderate degree of enlargement of the lymph nodes of the hili of the lungs. The patient was discharged from the hospital on 8 July.

CASE 2. A 31-year-old man was admitted to the surgical service of this hospital on 25 May 1953 with the chief complaints of a dull, aching pain in the posterior part of the right side of his chest and a tender swelling over the right ninth rib posteriorly of about 12 weeks' duration. A cough productive of a moderate amount of thick yellow sputum had been present for about 9 weeks.

The patient was well developed and well nourished. A tender mass about 3 cm in diameter was present in the right ninth rib about 10 cm to the right of the midline.

The hemoglobin was 88 percent and the white blood cell count was 12,800 per cu mm, of which 61 percent were neutrophils, 30 percent lymphocytes, 8 percent eosinophils, and 1 percent basophils. The circulating polymorphonuclear eosinophilic leukocytes were 706 per cu mm. Serum calcium, phosphorus, acid phosphatase, and alkaline phosphatase were within normal limits. The total serum protein was 7.6 grams (albumin 4.9 grams, globulin 2.7 grams) per 100 ml. The albumin-globulin ratio was 1.81 : 1.00. The Eagle flocculation test for syphilis was negative. The urinalysis was negative. Bence Jones protein was not present in the urine. The roentgenogram of the chest showed the lungs to be clear and an oval and radiolucent area at the junction of the inner third and the middle third of the posterior part of the ninth rib (fig. 2). The roentgenograms of the skull, vertebral columns, sacrum, pelvis, humeri, and femurs did not show lesions.

About 8 cm of the right ninth rib was resected on 4 June. The tumor was located at the center of the resected part.

A soft pale grayish pink tissue replaced the bone, occupied almost the entire marrow cavity, and nearly completely eroded the cortex on the pleural surface of the rib. Microscopic examination showed the tumor to be an eosinophilic granuloma.

**Figure 2 (case 2). Roentgenogram on 27 May 1953, showing an oval and radiolucent area at the junction of the inner third and middle third of the posterior part of the ninth rib.**

The patient was discharged from the hospital on 15 June. He returned for follow-up examination, on 20 July 1953, 20 January 1954, 26 August 1954, and

27 January 1955, and the roentgenogram of the chest on each occasion did not show a new lesion.

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# Sting by a Venomous Lionfish

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A NUMBER OF tropical marine fishes are known to be capable of inflicting serious wounds by means of their venomous spines. For example, stings by the stonefishes *Synanceja horrida* (Linnaeus) and *Synanceja verrucosa* Bloch and Schneider cause excruciating pain and marked swelling; in addition, systemic effects may be severe, and death has occurred in a number of cases.[1-5] Another fish which has been responsible for painful wounds similar at least superficially to those produced by the stonefish is *Pterois volitans* (and other species of the same genus), a scorpionfish which occurs over wide areas of the tropical Pacific and Indian Oceans. This fish (commonly known as the lionfish, turkeyfish, or zebrafish) is found in shallow water and is often encountered by skindivers (fig. 1). Although it is generally recognized that stings by the spines of this fish may have serious consequences,[6-9] adequate case histories have been lacking. The present report describes a case in which serious local and systemic effects occurred.

## CASE REPORT

The victim was a 38-year-old man in good physical condition. He encountered the lionfish while skin-diving in about 15 feet of water at Parry Island, Eniwetok Atoll, Marshall Islands. His right hand accidentally came in contact with the dorsal fin of the fish, and one spine entered the middle and another the ring fingers of the hand. An unsuccessful attempt was made to express blood from the puncture wounds. Pain appeared immediately at the site and became very severe by the time the victim had reached the shore (about 5 minutes). About 20 minutes after the incident the injured hand was immersed in very hot magnesium sulfate solution for about 15 minutes, but the intensity of pain continued to increase and soon involved the entire extremity. A subjective sensation that the fingers of the affected hand were fluttering was experienced, although this was not actually the case. One of us (S. E. L.) was called into the case a few minutes later. By this time the victim was stuporous and in a state of severe shock. The two fingers were swollen to about double their normal size, and the forearm and arm were also swollen. Blood pressure was 0/0 mm Hg; heart rate was about 10 per minute, and the patient's skin was cold and moist. Epinephrine was administered intravenously, and the pulse rate increased and blood pressure became perceptible. Epinephrine administration had to be

From Department of Pharmacology, University of Southern California, School of Medicine, Los Angeles, Calif., and Kirtland Air Force Base, New Mexico.

repeated at intervals over a period of several hours in order to maintain blood pressure; at the end of this period the patient was fully conscious and pulse rate and blood pressure had returned to normal.

**Figure 1: Specimen of *Pterois volitans* (Linnaeus) from Eniwetok Atoll, Marshall Islands (standard length 24 cm). The venomous long dorsal spines (arrow) and the zebra-like markings on the body of the fish are apparent.**

Subsequent treatment included the intravenous administration of 500 mg of ascorbic acid, and the intramuscular administration of 100 mg of meperidine hydrochloride and 10 mg of diphenhydramine hydrochloride. An ice pack extending from fingertip to shoulder was applied overnight. The pain had disappeared by the following morning, and the patient was discharged from the hospital about 24 hours after the incident. The swelling of the fingers diminished only very slowly and a month had elapsed before they resumed normal size. The skin of both fingers eventually sloughed off. The victim has experienced no known after-effects during the 6 years following the incident.

## COMMENT

The local effects (very severe pain, swelling) seen in the case reported here are similar to those described previously;[8, 9] no reports of cardiovascular collapse have come to our attention, however. Recent studies on the pharmacologic actions in rabbits of the venom from the spines of *Pterois volitans* indicate that the primary effect of small doses is the production of hypotension, with essentially no change in the electrocardiogram.[6] Amounts of venom sufficient to lower the blood pressure to about one half also produce evidence of myocardial ischemia or injury (flattening or inversion of T wave, or displacement of S–T segment), which is reversible if the animal recovers. Injection of fatal doses produces effects initially which are similar to those described above; a variety of additional electrocardiographic changes soon appear (*e.g.*, extrasystoles, bundle branch block, ventricular tachycardia, ventricular fibrillation). The respiration slows and finally ceases, and the blood pressure continues to decrease. Initiation of artificial respiration in these rabbits immediately after respiratory arrest is ineffective in prolonging the life of the animal. Therapeutic measures directed toward the support of the circulation are suggested by these experiments, and the immediate response to epinephrine in the case reported here is in accord with this view.

The local effects experienced by victims of stings by lionfishes[6-9] and by stonefishes[1-5] (*e.g.*, excruciating pain, marked swelling) appear to be essentially the same. Furthermore, effects of the two venoms upon the cardiovascular system of the rabbit are also almost indistinguishable.[1, 2, 6] A further point of similarity is that the active substances in both cases are nondialyzable and apparently protein in nature.[1, 2, 6] These facts suggest that the active substance or substances in these venoms are closely related and that similar therapeutic measures should be used in both cases.

## SUMMARY

A case of severe poisoning following a sting by dorsal spines of the tropical marine lionfish (*Pterois volitans*) is reported. The victim experienced severe local pain and swelling, followed by cardiovascular collapse. Repeated administration of epinephrine was effective in restoring and maintaining blood pressure and heart rate. Local swelling persisted for a period of weeks after the sting, but no permanent after-effects were noted.

ACKNOWLEDGMENT: The details of the case were obtained in the course of an investigation of the venoms of various marine fishes; this investigation was aided by a contract between the Office of Naval Research, Department of the Navy, and the University of Southern California, NR 107-342. We wish to thank Dr. C. C. Custer of Lanai City, Hawaii, for furnishing some of the details of the medical treatment in this case.

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## "YES, DOCTOR PERRY"

There is an occupational hazard in the practice of medicine that, if not guarded against, can do the individual doctor, the profession as a whole and society at large a great deal of harm. It is the temptation for doctors to think of themselves as individuals set apart from the rank and file of humanity, set apart by knowledge, skill and power—and intellectual elite. It is easy to see how the temptation arises. Patients ask questions. Doctors give the answers. Patients do what we tell them to. Somehow we get to feel that we are entitled to an acquiescent, "Yes, doctor," from the whole human race on any matter whatsoever. We fall, in other words, into what the theologians call the sin of pride. We tend, like the Pharisee, to thank God we are not as other men are.—RALPH PERRY: I Have My M.D.—What Now? *Missouri Medicine*, August 1959.

# Military Medical News

**TRACHOMA VACCINE.** Following the announcement in November of a trachoma vaccine developed at the Navy medical research unit in Taipei, Taiwan, Dr. Edward H. Cushing, Deputy Assistant Secretary of Defense (Health and Medical), visited 3 of the doctors responsible for the vaccine. Shown from left to right are : Dr. Cushing; Dr. S. P. Wang, Taipei, a staff member of the research unit; Dr. J. Thomas Grayston, assistant professor of medicine at the University of Chicago, and presently attached to the research unit staff; and Captain Robert A. Phillips, MC, USN, commanding officer of the unit. Others who participated in the work on the vaccine included R. L. Woolridge, immunologist, Lake Bluff, Illinois, and P. B. Johnston, Chicago, civilian members of the research unit staff, and Dr. Y. F. Yang, professor of ophthalmology at the National Taiwan University College of Medicine.

## AFIP OFFERS EYE COURSE

An ophthalmic pathology postgraduate short course for military ophthalmologists who are board qualified or certified, or in their final year of residency training, and a limited number of civilian physicians, will be given at the Armed Forces Institute of Pathology 28 March to 1 April 1960. Subjects will include a review of the normal histology of the eye and changes incident to growth and aging; acute, chronic, and granulomatous lesions and their sequelae; injuries; cataract; glaucoma; vascular diseases; intraocular tumors; optic nerve pathology; and epibulbar and orbital inflammatory and neoplastic lesions.

## HOLDING VD SYMPOSIUM

The 11th annual symposium on recent advances in the study of venereal diseases will be held 7–8 April 1960, in Chicago. Sessions, at the Palmer House, will be open to all physicians and workers in allied fields interested in venereal disease. The symposium is sponsored by the American Venereal Disease Association and the U.S. Public Health Service.

## ORAL PATHOLOGY COURSE

A postgraduate short course on pathology of the oral regions for the general dentist and pathologist has been announced by the Armed Forces Institute of Pathology, 14–18 March 1960. Consisting of lectures, presentations of case histories, and conferences, the course will cover clinical and histopathologic features of inflammatory and neoplastic diseases of the lip, tongue, floor of mouth, cheeks, palate, and oropharynx; tumors of the odontogenic apparatus and cysts of the oral regions; pathology of the pulp and periodontium; normal embryogenesis of the teeth and jaws, and anomalies of the teeth with respect to size, shape, and development; and correlation of the oral manifestations of systemic disease with their histopathology, with equal emphasis on the clinical, histopathologic, and roentgenographic features of oral disease.

## *Official Decorations*

The following awards were recently announced by the Departments of the Army, Navy, and Air Force.

**Distinguished Service Medal**

Wilford H. Hall, Maj. Gen., USAF, MC

**Legion of Merit**

James M. Davis, Col., USAF, MC
Frederich J. Frese, Jr., Col., USAF, MC*

**Army Commendation Ribbon with Metal Pendant**

Leo A. Jachowski, Comdr., MSC, USN
James H. Mackin, Col., MSC, USA
Byron Glen McKibben, Col., MC, USA
H. Haskell Ziperman, Lt. Col., MC, USA

**Navy Commendation Ribbon with Metal Pendant**

Robert C. Bornmann, Lt., MC, USNR

**Air Force Commendation Medal**

Samuel A. Terris, Lt. Col., USAF, DC

*First oak leaf cluster

**AIR FORCE CHIEF NURSE.** Lieutenant Colonel Dorothy N. Zeller, USAF, NC, former deputy, succeeded Colonel Frances I. Lay, USAF, NC, as chief of the Air Force Nurse Corps on 8 January 1960. Colonel Lay has been named command nurse for the U.S. Air Forces in Europe, Wiesbaden, Germany. A 1935 graduate of the Philadelphia General Hospital School of Nursing, Colonel Zeller has served on continuous active duty for 23 years.

## *Officers Certified By Specialty Boards*

The surgeons general of the Navy and Air Force have announced that the following regular Medical Corps and

Veterinary Corps officers have been certified by the specialty boards indicated:

**American Board of Plastic Surgery**

O'Brien, Robert W., Comdr., USN

**American Board of Surgery**

Cales, Robert J., Lt. Comdr., USN

**American Board of Veterinary Public Health**

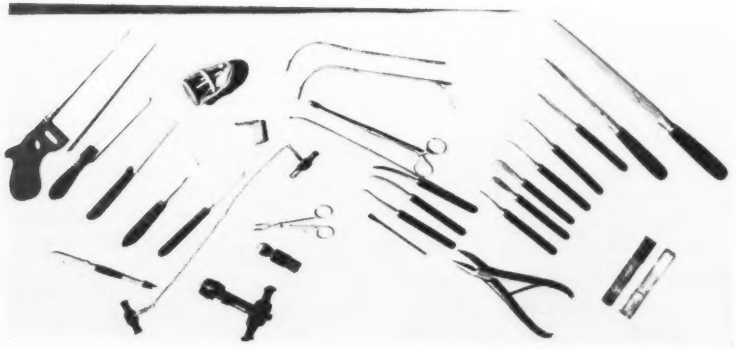
Miller, Robert R., Col., USAF

## *DEATHS*

BURKE, HERBERT JAMES, Captain, MSC, USAR, of Belleville, Illinois; assigned to the Office of the Army Surgeon General, Supply Division; graduated in 1940 from Oklahoma School of Accounting; served in enlisted status 3 April 1941 to 29 May 1945; commissioned as a second lieutenant in the Army of the United States 30 May 1945; died 25 November 1959, aged 41, at DeWitt Army Hospital, Fort Belvoir, Virginia, of a heart attack.

HANSEN, HELEN MARIE, Lieutenant Commander, NC, USN, of Minneapolis, Minnesota; assigned to U.S. Naval Hospital, San Diego, California; graduated in 1934 from Lutheran Deaconess School of Nursing, Minneapolis; commissioned as an ensign in the United States Navy 23 June 1942; reported for active duty 24 June 1942; died 22 October 1959, aged 47, at the U.S. Naval Hospital, San Diego, of brain stem syndrome.

MISCEVICH, MILDRED MITZI, Captain, AFRes, NC, of Versailles, Pennsylvania; assigned to 836th Tactical Hospital, Langley Air Force Base, Virginia; graduated in 1947 from McKeesport Hospital School of Nursing, McKeesport, Pennsylvania; appointed a first lieutenant in the Air Force Reserve and reported for active duty 6 August 1951; released from active duty 25 September 1954; recalled to active duty 11 May 1958; died 7 November 1959, aged 34, at Langley Air Force Base, of asphyxiation.



**CIVIL WAR INSTRUMENTS.** These surgical instruments, in use during the Civil War, are from the collections of the Medical Museum of the Armed Forces Institute of Pathology, which celebrates its 100th anniversary in 1961 concurrently with the Civil War Centennial. Colonel Frank M. Townsend, USAF, MC, director of the Institute, has been invited to serve on the Advisory Council of the Civil War Centennial Commission.

# Progress Notes

Brigadier General **Floyd L. Wergeland,** MC, USA, executive director, office for dependent's medical care, office of the Army surgeon general; Rear Admiral **Edward C. Kenney,** MC, USN, deputy and assistant chief of the Bureau of Medicine and Surgery; and Colonel **Aubrey L. Jennings,** USAF, MC, director of professional services, office of the Air Force surgeon general, represented the surgeons general of their respective services in the House of Delegates at the American Medical Association's clinical meeting in Dallas, Texas, 1–4 December 1959 . . . Lieutenant Colonel **Edward C. Knoblock,** MSC, USA, chief of the department of chemistry at Walter Reed Army Institute of Research has been promoted to his present rank . . . In imitation of civilian dental practitioners who use soft music in offices and operating rooms to soothe their patients, the dental department of the Naval Air Station at Jacksonville, Florida, has installed 21 speakers in the department, which provide a 20-minute flow of soft music from tapes, followed by a 4-minute period of rest. Captain **Macy G. Martin,** DC, USN, is the senior dental officer at the station.

The new dental intern program at Fort Knox, Kentucky, for Army dental officers entering the service, is under the direction of Colonel **Henry B. Fitch,** DC, USA . . . Captain **Robert A. Freyling,** MC, USN, U.S. Naval Hospital, Camp Pendleton, California, has been named executive officer and chief of orthopedics at the U.S. Naval Hospital, Corpus Christi, Texas . . . Colonel **Adam J. Rapalski,** MC, USA, has been named as commanding officer of the Army's Environmental Health Laboratory, Army Chemical Center, Edgewood, Maryland, replacing Colonel **Edward J. Dehne,** MC, USA, now preventive medicine officer, Second Army, Fort Meade, Maryland . . . Lieutenant Commander **Virginia Lee Riley,** NC, USN, an accomplished artist who studied at the Boston Museum of Art, recently exhibited a collection of her watercolors in Washington, D.C. Her work was also exhibited at the 22d annual Metropolitan Art Exhibition at the Smithsonian Institution, and at the Naval Medical Center, Bethesda, Maryland, where she is now stationed, during the 51st anniversary of the Navy Nurse Corps.

Captain **Shakeeb Ede** MC, USN, who succeeded Captain **Russell H. Blood,** MC, USN, as executive officer, will also serve as chief of surgery of the U.S. Naval Hospital, Great Lakes, Illinois. Captain Blood has been reassigned as Fourth Naval District medical officer, Philadelphia . . . Major **John E. Reisner,** MC, USA, is the new assistant chief of the dermatology service at Fitzsimons Army Hospital, Denver . . . Lieutenant **John D. Culberson,** MC, USNR, of the U.S. Naval Hospital, Philadelphia, has been appointed as an instructor in the department of pathology, Woman's Medical College of Pennsylvania . . . Colonel **George F. Lull,** MC, USA, former chief of diagnostic radiology at Fitzsimons Army Hospital, is now chief of radiology service at William Beaumont Army Hospital, El Paso, Texas . . . Captain **Gerald W. Hurst,** MC, USN, U.S. Naval Hospital, Great Lakes, Illinois, has been elected as a diplomate of the American Board of Otolaryngology . . Captain **Frederick A. Zehrer,** MSC, USA, formerly at Letterman Army Hospital, has been named to head the newly-created office of educational services, Army Medical Service School.

# The Medical Officer Writes

## Articles Published in Other Journals

### *U.S. Army*

CENTRALIZED FOOD SERVICE VERSUS DECENTRALIZED FOOD SERVICE. Major J. C. Lydon, AMSC, USA, Madigan Army Hospital, Tacoma, Washington. *Military Medicine*, August 1959.

GASTROSCOPY AND ESOPHAGOSCOPY: AN ANALYSIS AND DISCUSSION OF 1,752 PROCEDURES. Colonel B. H. Sullivan, Jr., MC, USA, and Captain J. E. Myers, Jr., MC, USA, Walter Reed Army Hospital. *Medical Annals of the District of Columbia*, August 1959.

USE OF SUCTION IN CONTROL OF RESIDUAL, INFECTED, OR DRAINING CAVITIES, TRACTS, AND FISTULAS. Captain K. Q. Vandenbos, USAF, MC, Lieutenant Colonel C. W. Hughes, MC, USA, and Colonel W. F. Bowers, MC, USA, Tripler Army Hospital, Hawaii. *A.M.A. Archives of Surgery*, June 1959.

CONGENITAL MIDGUT VOLVULUS: CASE REPORT WITH AUTOPSY. Captain W. J. Warwick, MC, USA, and Captain S. R. Leavitt, MC, USA, DeWitt Army Hospital, Fort Belvoir, Va., *Archives of Pediatrics*, August 1959.

OBSERVATIONS OF THE CARDIOVASCULAR SYSTEM IN EXPERIMENTAL TRAUMATIC SHOCK. Captain H. M. Wise, Jr., MC, USA, First Lieutenant A. T. Knecht, Jr., CmlC, USA, PFC G. S. Beals, USA, and PFC M. Yessis, USA, Army Chemical Center, Edgewood, Maryland. *Surgery*, September 1959.

### *U.S. Navy*

THE UNRELIABILITY OF PAIN AS A SYMPTOM OF DENTAL PATHOLOGY. Lieutenant N. D. Sperber, DC, USNR, U. S. Naval Training Center, San Diego, Calif. *Journal of the American Dental Association*, September 1959.

CHANGING CONCEPTS OF DEAFNESS. Commander G. W. Taylor, MC, USN, U.S. Naval Hospital, Bethesda, Maryland. *Medical Annals of the District of Columbia*, July 1959.

EXTERNAL OTITIS: DIAGNOSIS AND TREATMENT. Lieutenant B. M. Webb, MC, USN, and Lieutenant J. E. Hansen, MC, USN, U.S. Naval Hospital, Bethesda, Maryland. *Medical Annals of the District of Columbia*, July 1959.

ELECTRONICALLY CONTROLLED CORONARY ARTERIOGRAPHY. Lieutenant H. C. Urschel, Jr., MC, USNR, and E. J. Roth, M.D., Naval Medical Research Institute, Bethesda, Maryland. *Annals of Surgery*, August 1959.

### *U.S. Air Force*

HUMAN ASPECTS OF MID-AIR COLLISION PREVENTION. A. F. Zeller Ph.D., U.S. Air Force Directorate of Flight Safety Research, Norton Air Force Base, Calif. *Aerospace Medicine*, August 1959.

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Titles of articles in current periodicals written by commissioned officers and civilian scientists in the medical services of the Department of Defense will be published each month insofar as space permits. This listing is gleaned from available periodicals and is not necessarily complete. Authors desiring inclusion of published articles are invited to send a reprint to the *Journal*.—EDITOR.

# This Is Your A.M.A.

THE AMERICAN MEDICAL ASSOCIATION organized its Council on Industrial Health in 1937, but had taken an active interest in industrial health for at least a quarter-century before that. As early as 1915 the Association's Section on Preventive Medicine and Public Health had a Committee on Industrial Sanitation, and the Association's Judicial Council had prepared an extensive review of workmen's compensation laws and their medical implications.

The scope and activities of the Council on Industrial Health have been expanded considerably over the years to keep pace with advances in industrial medicine. The Council has recently been assigned primary responsibility within the Association for space medicine and has delegated this to its Committee on Aviation Medicine.

However, the Council is concerned principally with the multitudinous problems connected with the protection and improvement of the health of the working population. The Council advocates medical examinations and supervision of all workers to ensure suitable job placement. It encourages health maintenance through health education and counseling, periodic examinations, and prevention and treatment of occupational illness and injury.

Other areas of interest and activity of the Council include workmen's compensation, rehabilitation, employment of the handicapped, vision and hearing conservation, and investigation and control of all health hazards in industry, and the promotion of cooperation in these areas among physicians, industrial hygienists, and nurses. The Council promotes the teaching of occupational medicine at all levels of medical education, and encourages and assists physicians in other fields of practice to learn more about the health of the worker in relation to his job.

The Council on Industrial Health serves as a source of information and guidance in all of the above as well as in the following areas: the organization, operation, staffing, and the proper scope, objectives, and functions of occupational health programs; the practice of occupational medicine and its relationships with management, labor, and medicine in general; and the training, qualification, board certification, and employment opportunities in occupational medicine.

The Council carries out its work primarily with the advice and help of 5 Standing Committees and 11 Subcommittees. The Standing Committees are: Committee on Aviation Medicine, Committee on Interprofessional Relations, Committee on Medical Education and Training, Committee on Public Services, and Committee on Scientific Development. The Subcommittees are: Committee on Industrial Nursing, Committee on Mental Health in Industry, Committee on Industrial Health Emergencies, Committee on Medical Care for Industrial Workers, Committee on Workmen's Compensation, Committee on Occupational Cancer, Committee on Occupational Dermatoses, Committee on Industrial Ophthalmology, Committee on Industrial Medical Records, Committee on Neurological Disorders in Industry, and Committee on the Pneumoconioses.

These committees have developed pamphlets on a large number of occupational health subjects. Single copies of these pamphlets may be obtained free by writing to the Council on Industrial Health, American Medical Association, 535 North Dearborn Street, Chicago 10, Illinois. Among the titles are:

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From the Council on National Defense of the American Medical Association. The views and opinions expressed are not necessarily those of the Department of Defense.—EDITOR.



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Scope, Objectives and Functions of Occupational Health Programs
Guiding Principles of Medical Examinations in Industry
Guiding Principles and Procedures for Industrial Nurses
The Legal Scope of Industrial Nursing Practices
The Survey of the Occupational Environment
Tonometry in Glaucoma Surveys
Threshold Limit Values for 1959
Occupational Cancer—Methods of Epidemiological Study
Occupational Dermatoses
Medical Relations in Workmen's Compensation
A Guide for Medical Services for Construction Projects
Syllabus on Work Absence
Compulsory Temporary Disability Insurance Programs

The Council stands ready to comply with requests for advice and assistance from members of the Armed Forces on occupational health problems. On the Council and its committees are several active and many retired members of the Armed Forces and effective liaison is maintained between the Council and the offices assigned responsibility for industrial health in the Army, Navy, and Air Force. Representatives of these offices have attended many of the meetings of the Council. The Council recently reviewed an Air Force film entitled "Air Force Industrial Medicine in Action" and initiated action to promote its wider use. The Council hopes that this liaison will continue and grow in effectiveness.

## Monthly Message—continued from page ii

Concurrently various guest lecturers are available to the schools. For example, the Atomic Energy Commission has offered members of its staff and consultants.

Although the funding of the program was on a very uncertain foundation in its early years, the broad acceptance of the program and the participation of the Department of Health, Education, and Welfare, and the Office of Civil and Defense Mobilization has made the program more stable. During the school year 1958–1959, 55 schools participated in the program and 70 during 1959–1960.

Many letters have been received both in this office and the Office of the National Coordinator complimenting the program and the willingness of the military services and affiliated agencies to share their work with the medical schools. Personally, I believe this is one of the more important functions of military medicine in its relation with our medical schools and the civilian profession.

Frank B. Berry

FRANK B. BERRY, M.D.
*Assistant Secretary of Defense*
*(Health and Medical)*

# Correspondence

St. Louis, Missouri

I think the redesigned cover and format is most attractive and I want to congratulate you as editor and your editorial staff and board for the excellent job you are doing.

STANLEY F. HAMPTON, M.D.

College of American Pathologists
Tacoma General Hospital
Tacoma, Washington

I have been privileged to receive and read the Armed Forces Medical Journal for the past ten years. I have been extremely pleased with the progress made in the betterment of the Journal. The quality of articles has consistently improved as well as the general format of the Journal. I would place the Armed Forces Medical Journal high on the list of recommended reading material not only for Army and Reserve Officers, but for any general practicing physician. On the occasion of the tenth anniversary of the reorganized United States Armed Forces Medical Journal publication, I wish to compliment you and all of the others of the editorial board not only for having maintained the high quality of the Journal but also for having instituted innovations that have made the Journal more valuable to its readers.

CHARLES P. LARSON, M.D., PRESIDENT

Temple University Hospital
Philadelphia, Pennsylvania

I note with pleasure the new format of the U.S. Armed Forces Medical Journal. This is in fitting anticipation of the tenth anniversary of its appearance as the professional publication for the medical components of the Department of Defense. I could expound at length on the many fine features and the high and rising standard of excellence of its content, but I think it can be summed up by saying that the Journal has taken its place in the front ranks of the medical publications of the world.

RICHARD A. KERN, M.D.

# Book Reviews

BEHAVIOR PROBLEMS OF CHILDREN IN NAVY OFFICERS' FAMILIES: as related to social conditions of Navy family life. A dissertation by *Genevieve Gabower*, B.A., M.A. 279 pages. Catholic University of America Press, Washington, D.C., 1959. Price $3.

This paper-bound book, a dissertation submitted by the author in partial fulfillment of requirements for the degree of doctor of social work, demonstrates considerable effort. The author has undertaken to study the effects of problems peculiar to naval life on the emotional development of naval children. She bases her work on a 2-year period as psychiatric social worker at the U.S. Naval Hospital, Bethesda, Maryland, where she had an opportunity to interview children who had come to the attention of the hospital psychiatric service, as well as their parents. A group of 15 children of regular naval officers, selected from a total of 43 who had received psychiatric treatment during the calendar year 1956, were paired with a control group of children who had never required psychiatric attention. From this study the author has arrived at many well stated conclusions: The fathers of the behavior group had had less schooling than those of the control. The mothers of the behavior group had been older at the time of their marriage. One fifth of the parents of the behavior cases thought that moving had been a disadvantageous influence upon their child's behavior; none of the parents of controls believed this. Behavior cases were found to have had more separations from the father from infancy through adolescence. Parents of behavior cases were less active in identifying themselves with the civilian community in which they lived. This work should help in the understanding of conditions which lead to good emotional development in childhood. It will be of interest to students of social case work because the author has painstakingly gone through all the steps necessary in securing a good case history; to naval personnel; and to those unfamiliar with military procedures.

CAPT. FRANCES L. WILLOUGHBY, MC, USN

TRAUMA, by *Harrison L. McLaughlin*, M.D. 784 pages; illustrated. W. B. Saunders Co., Philadelphia, Pa., 1959. Price $18.

This fine volume reflects the guiding hand of the author, who has utilized the assistance of 19 experienced representatives of the surgical specialties. Much of the material presented is of orthopedic significance, but this text transcends that specialty, embracing the wider aspects of trauma. Emphasis on first things first is clear at the outset, and the book is not clogged with inconsequential, all-inclusive minutiae, but dwells at length on common traumatic entities which are at times so plaguing to treat. For example, the masterful presentation of the whole aspect of Colles' fractures leaves little wanting. There are 5 main sections to the book, the middle 3 being primarily concerned with regional trauma. The opening and closing sections on general considerations and on the head and special senses make this an outstanding work of interest to all dealing with trauma. The emotional response to injury is concisely set down without a jumble of semantics, and is an admirable common-sense exposition. Biochemical and

physiologic principles related to injury and healing are presented with lucidity, giving the reader a grasp of the metabolic response to injury unclouded by needless and confusing graphs. Whatever graphs and tables are employed are comfortingly simple. Well-selected roentgenograms are augmented by clear drawings which leave little doubt as to the points made. Anatomic drawings are good, though rather sparse in some sections. Especially outstanding is the chapter dealing with injuries of the skull and brain. Precise recommendations for managing the patient with head injuries are given and, as is the pattern throughout the book, controversial discussions are kept to a minimum. The author states at the outset that this book was written not so much "for the edification of other surgeons as for the innumerable medical students, residents, and practitioners who will not receive a complete training in surgery, but who, nevertheless, will be called upon to cope with the great majority of all civilian accidents." He is too modest in his estimate of the scope of his audience. With the specialization existing today, no one surgeon dealing with trauma can know all the answers, and he will certainly be grateful for an authoritative, practical, and well-written volume to help him channel his thinking with respect to the care of the severely injured patient. Any dogmatism present is inoffensive. This book should be readily available to all who treat trauma. COMDR. ROBERT H. BROWN, MC, USN

THE YEAR BOOK OF DERMATOLOGY AND SYPHILOLOGY (1958–1959 Series), edited by *Rudolf L. Baer*, M.D., and *Victor H. Witten*, M.D. Year Book Publishers, Inc., Chicago, Ill., 1959. Price $8.50.

This annual volume contains abstracts of the articles the editors consider of most significance in the foreign and American dermatologic literature published between September 1957 and September 1958. Each abstract contains the author's name, the name of the facility in which the work was done, and the journal in which the article was published. Following each abstract or series of similar abstracts there is a note of editorial opinion and experience on the subject. The articles are grouped under 14 sections, the first section being devoted to a review of benign pigmented lesions. The volume is well indexed and serves as a quick and ready reference for the busy dermatologist.

CAPT. KARL V. KAESS, MC, USN

SURGERY OF THE COLON, by *E. S. R. Hughes*, M.D., M.S. (Melb.), F.R.C.S. (Eng.), F.R.A.C.S. 415 pages; illustrated. Williams & Wilkins Co., Baltimore, Md., 1959. Price $12.50.

This book records the author's personal ideas and experiences with diseases of the colon. Because he decries dividing the management of colonic disease into medical and surgical categories, he includes chapters on constipation and diarrhea, in neither of which is surgical treatment recommended. Neither Hirschprung's disease nor appendicitis is discussed, the former omitted becasue of the author's lack of experience with the disease and the latter because of the "unique position it occupies." The volume is, in the main, orthodox and well written, but there are areas in which it is neither current nor orthodox according to American standards. For example, antibiotic preparation of the bowel for surgery is viewed with mild lack of favor, as is the use of blood in amounts of 1 unit on the day before surgery in preparation of the patient because the increased blood volume thus obtained would increase surgical bleeding. Operative technics, when mentioned, differ from ours in detail, but the general principles are according

to our standards. The book is well organized, the presentation interesting. The book is printed on heavy paper of good quality and most of the illustrations are simple, lucid sketches which add significantly to the clarity of the text. A chapter rarely seen in a book on colonic lesions is the one on mesenteric vascular disease. It is unusual and rather well presented, though more of theoretic interest than of practical value. This book would be worth the purchase price only to one interested in having the most complete library possible on the subject of surgery of the colon.

COL. DON S. WENGER, USAF, MC

THE YEAR BOOK OF PATHOLOGY AND CLINICAL PATHOLOGY (1958–1959 Year Book Series), edited by *William B. Wartman*, B.S., M.D. 502 pages; illustrated. Year Book Publishers, Inc., Chicago, Ill., 1959. Price $8.50.

As expected, William Wartman and his editorial staff have again succeeded in highlighting the year in pathology and clinical pathology in such a way as to give the reader a very comprehensive view of the important advances in the field. As in the past, summaries of important articles with editorial comment are arranged under major headings relating to organs or systems, making it easy for the reader to find information relating to a specific subject. One hundred years have passed since the death of Richard Bright, and the editor has seen fit to include a very appropriate editorial on this famous physician. A special article, "A Century of Cellular Pathology" by Sir Roy Cameron of London, is also included. The section on clinical pathology is most timely, presenting summaries of articles on all of the new major testing methods and theories. Of particular note is the section on microbiology, whose editorial note entitled "What Price Cleanliness?" emphasizes a lesson that seems so hard for the medical profession to learn. In keeping with its tradition the publisher has used a good binding in a "pocket size edition" with good reproduction of illustrations, and easily read print. This volume has certainly accomplished its purpose and is highly recommended.

CAPT. BRUCE H. SMITH, MC, USN

PEDIATRIC CLINICS OF NORTH AMERICA, May 1959. Symposium on Neoplastic Diseases. Vol. 6, No. 2. *James B. Arey*, M.D., consulting editor. 317 pages; illustrated. W. B. Saunders Co., Philadelphia, Pa., 1959. Price $15 per set of 4 books issued quarterly.

This concise book presents a symposium on neoplastic diseases of infancy and childhood. The information represents the most recent thinking as a cross section from this country and England (two contributors). All the various aspects —statistics, symptomatology, diagnosis, prognosis, and treatment—are discussed by the contributors. That all areas of the subject are covered is attested to by the fact that, of the 25 contributors, 3 are pediatricians, 8 are pathologists, 6 are surgeons, 4 are radiologists, 2 are internists, 1 is an ophthalmologist, and 1 is a neurosurgeon. In 17 chapters, the authors discuss the important clinical forms of neoplasms affecting the various parts of the body and organ systems. The chapters on tumors of the central nervous system, neuroblastoma, renal tumors, management of hemangiomas in infants, teratomas, and leukemia and lymphoma in children are of especial interest. An interesting new aspect in the treatment of neuroblastoma is reported by Dr. Bodian of the Hospital for Sick Children, London, England. A group of 42 children with neuroblastoma were treated with vitamin $B_{12}$, with clinical remissions in the majority of those under 1 year, and only small success in those over 1 year. Better survival rates in infants as

compared with older children have already been recognized. Therefore, the editor comments that until further data are available it might be well to note Dr. Bodian's conservative approach: "No patient has been denied the possible benefits of surgery and/or radiotherapy." This book should be of great value to anyone who deals with neoplastic diseases of children, including pediatricians, pathologists, radiologists, and surgeons.

COL. CONN L. MILBURN, JR., MC, USA

THAT THE PATIENT MAY KNOW, An Atlas for Use by the Physician in Explaining to the Patient, by *Harry F. Dowling*, M.D., Sc.D., and *Tom Jones*, B.F.A., assisted by *Virginia Samter*. 139 pages; illustrated. W. B. Saunders Co., Philadelphia, Pa., 1959. Price $7.50.

If the saying "one good picture is worth a thousand words" is true, then this book with its many excellent drawings should do much to preserve the medical larynx, while improving the understanding of the patient. It is a systematized collection of anatomically accurate drawings of both normal structures and their lesions, labeled in simple terms, to be used by the physician in his discussion of a problem with his patient. Numerous schematic drawings of physiologic processes are presented to aid in the elaboration of metabolic, endocrine, and psychologic factors in disease. It is doubtful, however, that a drawing of a throbbing foot will convey any additional information to one afflicted with the neuritis of vitamin deficiency, or for that matter with any other ailment. Nor is such a loaded term as "shriveled" in the valvular heart disease section without hazard in these times of subliminal persuasion. Such objections, of which I have mentioned two, are of minor import and should not frustrate the mission of those who take the degree of doctor literally. Suggested drawings are available by area and disease by means of a good cross index. To many a busy practitioner this book may be a useful labor-saving device, yet it must be realized that a complex, dynamic problem does not become simpler merely because an artifactitious caricature of it has made it more easily transmitted to others.

LT. COMDR. ROBERT E. DE FOREST, MC,USN

MEDICINAL CHEMISTRY, A Series of Reviews Prepared Under the Auspices of the Division of Medicinal Chemistry of the American Chemical Society, by *Wilbur J. Doran*. *F. F. Blicke* and *R. H. Cox*, editors. *L. A. Woods*, associate editor. *Harriet Geer*, assistant editor. Vol. IV. 334 pages. John Wiley & Sons, New York, N.Y., 1959. Price $12.

The volume is principally devoted to tabular summaries of barbituric acid derivatives (classified according to structure) and of the available pharmacologic data pertaining to those compounds. This information is based upon a literature search that included over 1200 references found in *Chemical Abstracts* (1907 through 1956) and in *Chemische Zentralblatt* (1897 through 1944, except for the years 1940 and 1943), as well as a review of more than 200 barbituric acid derivatives tested at the I.-G. Farbenindustrie plant, in Germany. A relatively short introductory chapter provides summaries of a wealth of understandings concerning the chemistry, pharmacology, and animal metabolism of the barbituric acid derivatives. The same chapter provides brief discussions of potentiation of and antagonism to barbiturate depression, and of tolerance, habituation, chronic intoxication, and addiction to the barbiturates. Tabulation of the extensive data follows a scheme according to which the barbiturates are divided into three main classes, dependent upon the absence or presence of substitution on the nitrogen

(classes I and II: 5- substituted; 1- and 1, 3- substituted barbituric acids), and the thiobarbituric acids (class III). Most of the associated pharmacologic findings pertain to the activity observed in white rats or mice. This compilation of information can be expected to be invaluable to those workers for whom it is principally intended—the chemists and pharmacologists who are concerned with the synthesis and evaluation of new barbiturates. The forensic toxicologist will be much interested in the introductory chapter and will welcome the tabulations of the barbituric acid derivatives by generic and trade names. All who use the volume will be most appreciative of the clarity of the print, the distinctness of the structural formulas, and the ease achieved in the handling of the many literature references.

COL. THADDEUS J. DOMANSKI, USAF, MSC

A MANUAL OF BANDAGING, STRAPPING AND SPLINTING, by *Augustus Thorndike*, M.D., F.A.C.S. 3d edition. 153 pages; illustrated. Lea & Febiger, Philadelphia, Pa., 1959. Price $2.75.

This is a simple manual prepared for nurses, interns, and medical students, by a surgeon of wide experience. It may also benefit many physicians, for it covers a field notoriously inadequately taught in some medical schools. Suggestions for treatment appear but are rare. In the main, the essay is devoted to descriptions of the physical aspects of bandaging, strapping, and splinting. Dressings for each portion of the body are described and illustrated, with special emphasis being given to areas such as the head and hand, where the problem is a little more complicated. The discussion includes sections on sterile and septic dressings, plaster bandages, transportation of fractures, as well as material on sprains, strains, contusions, dislocations, and their bandaging. This manual occupies a position midway between a first-aid booklet and the more complex orthopedic texts. This little book would be of value to any physician who is being faced for the first time with the multiple problems of dressing cases of trauma and may well be especially valuable to the young military physician.

COL. DON S. WENGER, USAF, MC

DISEASES OF THE NOSE, THROAT, AND EAR, edited by *Chevalier Jackson*, M.D., Sc. D., LL. D., F.A.C.S., and *Chevalier L. Jackson*, M.D., M.Sc., F.A.C.S., with the collaboration of 61 outstanding authorities. 2d edition. 886 pages; illustrated. W. B. Saunders Co., Philadelphia, Pa., 1959. Price $20.

Any text produced by Chevalier Jackson is always outstanding. This edition presents improvements and advancements in the diagnosis and treatment of diseases of the nose, throat, and ear since the first edition was published, as well as continuing the essential basic information needed for a thorough understanding of these areas. The presentation is in an orderly, clear, concise manner. The material is divided in the same manner as in the original edition, namely, into parts: nose and nasal accessory sinuses; mouth, fauces and pharynx; ear; larynx and hypopharynx; and bronchi and esophagus. There is a chapter on anatomy and another on physiology for each of the areas treated. The various conditions are handled in separate chapters by various authors; some of these chapters have been entirely rewritten. The group of doctors who assisted in various areas give added authority to the presentation. Every otolaryngologist and bronchoesophagologist will want to be familiar with this edition. General practitioners and students will find it of even greater assistance than the previous edition.

COL. HENRY S. MURPHEY, MC, USA

THE MEANING OF POISON, by *Lloyd G. Stevenson*, M.D., 7th series, Logan Glendening. Lectures on the History and Philosophy of Medicine. 53 pages. University of Kansas Press, Lawrence, Kans., 1959. Price $2.

In the first of these two lectures Dr. Stevenson deals with the history of the various meanings given to the words *poison*, *contagion*, and *infection*. The primitive idea of the poison of a plant, drug, serpent, or even of a maiden was followed by such a term as our modern "blood poison" and by the concept of the toxins of contagious and infectious diseases. The second lecture traces the history of curare, the "hellish oorali" of Lord Tennyson, from its place as a center of controversy over antivivisection in the nineteenth century to its use as a research tool and adjunct of anesthesia at the present time. The little book is well bound and printed and has bibliographical notes and an index.

CAPT. LOUIS H. RODDIS, MC, USN (RET.)

YEAR BOOK OF MEDICINE, 1958–1959 Series, edited by *Paul B. Beeson*, M.D., *Carl Muschenheim*, M.D., *William B. Castle*, M.D., *Tinsley R. Harrison*, M.D., *Franz J. Ingelfinger*, M.D., and *Philip K. Bondy*, M.D. 782 pages; illustrated. Year Book Publishers, Inc., Chicago, Ill., 1958.

This is a practical clinical volume on the newer trends as published and selected throughout the period indicated. Although advances in therapy have been rapid and tremendous, particularly with antimicrobial, hypotensive, and steroid compounds, the authors have made a good selection of material. The sections on hematology and cardiovascular disease appear particularly comprehensive. The section on chest disease has two worthwhile articles on the pros and cons of BCG vaccination, bringing one up to date in this field. It would seem that the index is more inclusive than in some of the previous editions. This is a quick reference for the busy clinician and resident.

BRIG. GEN. FRANCIS W. PRUITT, MC, USA

PRINCIPLES OF DISABILITY EVALUATION, by *William Cauthorn Smith*, M.D. 210 pages. J. B. Lippincott Co., Philadelphia, Pa., 1959. Price $7.

This book on evaluation of physical disability provides a reference for physicians in industrial medical practice and appears to be of limited usefulness to others in the practice of medicine. It is published in large, easily read type with the contents divided into titled sections, subsections, and paragraphs. The book contains an excellent discourse on the medical witness, expert medical testimony, and courtroom deportment. The author is the chief medical advisor to the Oregon industrial accident commission, and the text repeatedly refers to the statutes of the state of Oregon and cautions the reader to familiarize himself with the laws and directives governing industrial disability evaluation and compensation in his own state. The author states that as a result of the marked lack of uniformity in industrial disability evaluation and compensation in the United States, many generalizations in the text are necessary. The text is replete with definitions of terms frequently misused and misunderstood in industrial medical practice. It occasionally becomes too wordy, especially when exploring the philosophy and history of disability evaluation. An excellent section is presented on examination of patients and on correlation of physical findings with job requirements and motivation of the worker. The book is well organized and contains a cross reference and index to make it more easily used.

LT. COL. FREDERICK S. SPIEGEL, USAF, MC

THE PRACTICAL EVALUATION OF SURGICAL HEART DISEASE, The Glover Clinic. Written and compiled by *Robert G. Trout*, M.D., edited by *Robert P. Glover*, M.D., *Joseph Sunner*, medical illustrator. Heart sounds recorded by *J. Scott Butterworth*, M.D. 132 pages; illustrated. Record of heart sounds inclosed. Blakiston Division, McGraw-Hill Book Co., Inc., New York, N.Y., 1959. Price $10.

This rather drastic simplification of the subject of surgical heart disease is an effort to bring to the reader in very simplified form the recent great advances in the therapy of congenital and acquired heart disease. The book is divided into four sections. The first section covers simplified physiology, roentgenographic interpretation, and electrocardiographic interpretation; the second section covers congenital and acquired pathologic conditions of the heart and great vessels; the third section covers diagnostic technics; and the fourth section contains the bibliography. In the second section, the clinical conditions discussed include a colored diagram of the heart and great vessels to show the altered blood flow, a diagram to indicate where the murmur is best heard, and pertinent roentgenograms and electrocardiograms. Pathologic physiology, association with other defects, symptoms, physical findings, prognosis with and without surgery, operative mortality, and the optimum age for operation are all listed in outline form. A typical case is presented for each condition discussed. Angiocardiography and right and left heart catherization are discussed briefly, along with appropriate diagrams of these procedures. The bibliography is adequate and up to date. Furnished with the book is a phonograph record of 9 common murmurs. This is extremely helpful to beginners and surgeons who find cardiac murmurs difficult to hear or evaluate. This is not a book for the cardiologist, but rather for the busy general practitioner or young surgeon who wants to bring himself up to date on the practical evaluation of surgical heart disease.

COL. SANFORD W. FRENCH, III, MC, USA

TEXTBOOK OF PHARMACOLOGY FOR NURSES, by *Margene O. Faddis*, R.N., M.A., assisted by *Joseph A. Hayman, Jr.*, M.D., B.A. 5th edition. 451 pages; illustrated. J. B. Lippincott Co., Philadelphia, Pa., 1959. Price $5.50.

This is a remarkably good textbook. All the desirable tenets of pedagogy are wedded with excellent publishing to produce a work that will live as happily ever after as any textbook can in the fluid and expanding field of pharmacology. The book is concise as it must be to be of value in the crowded curriculum of student nursing. And yet no important drug or group of drugs has been omitted from discussion. Such recent advances as tranquilizers and radioisotopes are well covered for the student nurse. Throughout the book there is a gracious and well-rewarding amount of physiology. Purposes of study and questions accompany each chapter and aid in shifting pharmacology from a rote memory chore to an intellectual understanding process. The mathematics of dosage are discussed in logical detail. The sections on insulin dosage and pediatric digitalis therapy are superb. The sections relating to technics or parenteral administration of drugs are adequate. The toxicology of each drug discussed is given ample stress, and various study plans throughout the book lay considerable emphasis on possible toxic reactions of therapy and the nurse's responsibility in early assessment of such reactions. This text is highly recommended for student nurses, for service corpsmen doing ward work, and as a pleasant refresher in pharmacology for graduate nurses.

CAPT. EDWARD J. HUYCKE, MC, USA

DIABETIC MANUAL, by *Elliott P. Joslin*, M.D., Sc. D. 10th edition. 304 pages; illustrated. Lea & Febiger, Philadelphia, Pa., 1959. Price $3.75.

This continues to be the outstanding manual on diabetes for the patient. Written in language any reader can understand, it should be required reading for all diabetic patients. The philosophy of Dr. Joslin on the education of the diabetic permeates every page. All general practitioners would profit from reading this book. Diet, exercise, prophylactic procedures, and complications are explained thoroughly, and there is an adequate index. The newer oral hypoglycemic drugs are discussed but not with much enthusiasm. The reference to Dr. Joslin's patients will give hope to those needing this book. The manual is highly recommended for all diabetic patients, but especially newly discovered cases, and at least one reading would also benefit those who care for diabetic cases.

CAPT. EDWARD P. MC LARNEY, MC, USN

MECHANISMS OF HYPERSENSITIVITY, Henry Ford Hospital International Symposium, edited by *Joseph H. Shaffer*, M.D., *Gerald A. LoGrippa*, M.D., and *Merrill W. Chase*, Ph. D. 754 pages; illustrated. Little, Brown & Co., Boston, Mass., 1959. Price $18.50.

This tremendous undertaking is a record of an international symposium sponsored in March 1958 by the Henry Ford Hospital in Detroit, Michigan. The material has been compiled and presented by 65 specialists from the United States, Canada, and several European countries with the cooperation of 450 investigators in the fields of allergy, dermatology, immunology, and allied sciences. The book is especially well organized and produced on fine-grade paper with excellent illustrations. The subjects are arranged into 12 major topics with discussion of each chapter by authorities in the representative fields. A detailed list of references also follows each chapter. The authors point out that antibodies cannot be divided into broad general classes on the basis of reaction with antigens because all antibodies can agglutinate, precipitate, and hemolyze. Some new hemagglutination methods for demonstration of antibodies in sera of ragweed-sensitive persons are described, namely, coupling of ragweed pollen constituents to rabbit red blood cells by stable covalent azo bonds and aggregation of "sensitized" erythrocytes by antibodies in allergic sera. The ideal antibody test should be able to detect specific antibodies in very small amounts, measure primary interaction of antibody and antigen, and not be dependent on a secondary reaction. It should provide quantitative data and "avidity," and biologic properties such as precipitating, nonprecipitating, and skin sensitizing. It should apply to any antibody-antigen system and be simple to perform. This ideal has not yet been reached. By electron microscopy red blood cells in hypersensitive states appear to change from the normally smooth surface to cones and umbilicated plateaus. This reaction has also been demonstrated in certain autoimmune diseases such as acquired hemolytic anemia, erythroblastosis fetalis, and anti-A and anti-Rh antibody reactions. Vascular spasm in pulmonary vessels is often considered the predominant factor in anaphylactic reactions; but it has been demonstrated in rabbits that the pulmonary capillaries are distended with eosinophilic or antigen-antibody precipitates and thrombi. These thrombi are also seen in renal glomeruli, splenic sinusoids, and some of the terminal portal veins of the liver. The book closes with a banquet speech entitled "AreWe Too Trigger-happy?" by A. Ashley Miles, M.D., of London, a philosophic and yet rather amusing and I believe important speech. This research-oriented work has excellent reference value for the clinician and laboratory officer as well as for the basic scientist.

COL. U. R. MERIKANGAS, MC, USA

ARTHRITIS, General Principles, Physical Medicine, Rehabilitation, edited by *Edward W. Lowman*, M.D., with 24 collaborators, foreword by *Howard A. Rusk*, M.D. 292 pages; illustrated. Little, Brown and Co., Boston, Mass., 1959. Price: $9.50.

This book, a compilation of the writings of 25 authors, serves as an up-to-date comprehensive review of the disorders collectively known as arthritides. Primary emphasis is given to the physiotherapeutic management of these important diseases. By way of preface, there is a brief discussion of anatomy, pathology, clinical aspects, and medicinal treatment in which the limitations of the latter are rightfully stressed. In addition to the organic forms of arthritis, there is included an excellent discourse on psychogenic rheumatism. The final three fourths of the volume concerns a discussion of how physical medicine and rehabilitative methods serve to benefit the arthritic. The gravity of the problem is well emphasized. Such methods as active-passive exercises, the usefulness and limitations of heat and massage, self-help devices, occupational therapy, the proper use of splints, and orthopedic surgical correction are thoroughly considered. One point worth noting is the impracticability of repeated and prolonged treatment in the hospital physiatric department. Most physiotherapy must be performed in the home by the patient himself with assistance from members of the family and, if possible, by a visiting nurse or mobile physical therapy unit. The volume is well illustrated with diagrams, photomicrographs, x-rays, pictures of patients, and self-help devices. Range of motion charts and detailed instructions for exercises are also included. It would appear that this volume should be of great reference value to any physician who attempts the management of patients with arthritis and especially important for the physiatrist.

CAPT. ROBERT J. WHIPPLE, MC, USN

PROCEEDINGS OF THE WORLD CONGRESS OF GASTROENTEROLOGY AND THE FIFTY-NINTH ANNUAL MEETING OF THE AMERICAN GASTROENTEROLOGICAL ASSOCIATION, Washington, D.C., 1958. Vol. I and II. 1,360 pages; illustrated. Williams & Wilkins Co., Baltimore, Md., 1959. Price $20.

The World Congress of Gastroenterology, held in Washington, D.C. in May 1958, was very well attended by physicians from all over the world. The substance of their contributions and deliberations is presented in these volumes. A few of the papers are in Spanish or French, but more than 95 percent of the text is in English. The book follows the organization used in presentations to the Congress: (1) symposia with several short papers on a specific subject by recognized authorities from different geographic locations, (2) panel discussions by these same authorities, and (3) original contributions by individual authors. Principal topics covered are peptic ulcer, cancer of the stomach, intestinal malabsorption, intestinal infections and infestations, effect of nutrition on the liver and pancreas, gastrointestinal physiology, and affections of the liver, pancreas, and biliary tract. Variation in epidemiology and clinical features of each disease as seen in different parts of the world are a major point in the discussion. This publication offers a unique opportunity to become acquainted with the opinions and experiences of physicians in other countries concerning diseases commonly seen in our daily practice. In some instances a wide divergence of opinion exists, and in many cases the most promising leads to newer approaches are disclosed in papers from abroad. These volumes will widen the horizon for its readers, and prove useful in any library.

COL. BENJAMIN H. SULLIVAN, JR., MC, USA

COLE & ELMAN'S TEXTBOOK OF SURGERY, by *Warren H. Cole*, M.D., with 49 contributing authors and consultants. 7th edition. 1,224 pages; illustrated. Appleton-Century-Crofts, Inc., New York, N.Y., 1959. Price $17.

This authoritative and up-to-date student textbook has 49 contributing authors and consultants, 36 of whom are full professors, 21 of these being department chairmen. The book opens with a concise chapter on surgical history, which is so often neglected. Next, the history and physical examination of the patient are considered, followed by a discussion of laboratory aids in diagnosis. After this there are 13 chapters on basic subjects such as inflammation, wounds, fluid balance, convalescence, anesthesia, shock, systemic reaction to trauma, burns, and the emergency room service. Five chapters deal with fractures and orthopedics, followed by detailed discussions of surgical diseases by organ and system. Military surgery is considered in a separate chapter, as is the matter of medical ethics and conduct. Gynecology and urology each are covered in separate chapters. The book is current, well written, and will take its place as the peer of the several available contemporary student textbooks of surgery.

COL. WARNER F. BOWERS, MC, USA

PATHOLOGY OF TUMOURS OF THE NERVOUS SYSTEM, by *Dorothy S. Russell*, Sc. D., M.A., M.D., F.R.C.P., LL. D., and *L. J. Rubenstein*, M.D., with a chapter on Tissue Culture in Relation to Tumours of the Nervous System, by *C. E. Lumsden*, M.D. 318 pages; illustrated. Williams & Wilkins Co., Baltimore, Md., 1959. Price $13.50.

With no pretensions to comprehensiveness, this volume is intended as an up-to-date discussion of a complex field, with emphasis on biologic aspects and on problems of interpretation. The term "tumor" has been interpreted in its strict sense, omitting granulomatous and parasitic lesions but including tumor-like maldevelopments such as teratomas, dermoid cysts, and craniopharyngiomas, as well as dysgenetic syndromes associated with tumor formation. Writing for practicing pathologists, for neurologists and neurosurgeons, and for postgraduate students, the authors avoid detailed descriptions of the more common lesions in favor of lengthier treatment of more difficult tumors. Generous use has been made of small clinically silent tumors found by chance at autopsy. After a general discussion of incidence and pathogenesis, the section on intracranial tumors proceeds through the meninges, reticular tissues including lymphomatous proliferations, blood vessels, neuroectoderm, pineal gland, retina and optic nerve, and neural components of the pituitary gland, with chapters on growth and dissemination of neuroectodermal tumors, on secondary neoplasms including pituitary adenomas that impinge on the brain, and on mechanical and circulatory changes produced by expanding intracranial lesions. The structurally similar tumors of cranial and spinal nerve roots and of peripheral nerves are described together, followed by a chapter on tumors of peripheral neurons. Each chapter concludes with a selection of references considered especially valuable by the authors, whose inclination is to favor more recent publications containing reviews of antecedent literature. Although this is not an atlas, it is noteworthy that the majority of entities discussed are illustrated by one or more photographs, either gross or microscopic or both; and these have been carefully reproduced. The authors admit to an element of bias, inevitable in any discussion of such controversial material as the essential cellular nature of peripheral nerve tumors or the relationship between capillary hemangioblastoma and angioblastic meningioma; but they have performed a commendable job in presenting the principal conflicting views before adopting their own nomenclature or classification for convenience of discussion.

The semantic confusion that exists in the terminology of nervous tumors is resolved in some cases by listing the more common synonyms of the term selected, as in the presentation of gliomas, and in others by a step-by-step comparison of the various classification schemes. A closing chapter on the contribution of tissue culture to nervous tumors includes a critical appraisal that some will find stimulating and others provocative.

MAY SHERMAN, M.D

DYNAMIC PSYCHOPATHOLOGY IN CHILDHOOD, edited by *Lucie Jessner*, M.D., and *Eleanor Pavenstedt*, M.D., with 15 contributors. 315 pages. Grune & Stratton, Inc., New York, N.Y., 1959. Price $8.75.

This book, a compilation of 12 papers, makes an excellent attempt to examine observations and studies from a wide range of clinical and experimental work with children, both normal and pathologic. It presents viewpoints which vary from systematic research, as exemplified by John Benjamin's controlled and long-lasting study on the use of prediction, to pure clinical reporting and interpretation, as demonstrated by Epstein's and Friedman's paper, describing the play of a psychotic boy. The book is less for general consumption, even among psychiatrists, but holds much for the psychiatrist and those in allied professions who have an interest in dealing with children. Much of what is included is very thought provoking. I would like to mention specifically the paper by Benjamin; the description of simultaneous analysis of mother and child, by Sperling; and the phenomenon of countertransference as elaborated by Proctor.

LT. COL. JAMES F. DONOVAN, MC, USA

LOW INTENSITY RADIUM THERAPY, by *Charles L. Martin*, E.E., M.D., F.A.C.R. and *James A. Martin*, M.D., F.A.C.R. 257 pages; illustrated. Little, Brown & Co., Boston, Mass., 1959. Price $12.50.

The Martins have finally been persuaded to compile their experiences in the handling of accessible carcinoma under one cover. This is a book long awaited by radiotherapists throughout the world. Fortunately, the publishers have spared no expense. The volume is liberally illustrated, well arranged, and printed in easily readable type. For more than 30 years, in Dallas, Texas, the Martins have been attaining results with superficial cancer that amaze their colleagues. Glimpses of this work have appeared in the literature in more than 22 articles since 1923. Fortunately, no major change has been necessary in their therapeutic method for many years, so that we have available for comparative study a complete and detailed account of cancer treated with radium alone. This is particularly important, since radical surgery for similar conditions is now in vogue. Their technic is, basically, the delivery of 6000 gamma roentgens in 7 days. This is accomplished by low intensity radium sources placed interstitially at 1 cm distances. The sources used have over-all lengths of 11, 27.7, 51, and 60 mm; contain 0.66, 1.33, 2.4, and 3 mg of radium sulphate; and have active lengths of 0.5, 1.5, 3.5, and 4.5 cm, respectively.

The clinical material is presented by means of case records of 94 individuals. The major areas of disease discussed are the skin, oral cavity, cervical node metastases, cervix, and breast. Well-prepared charts give survival experience which compares favorably with other large groups of similar cases. The book will be of interest to all radiotherapists.

COL. EDWARD M. DEYOUNG, MC, USA

# New Books

*Books received by the* U.S. ARMED FORCES MEDICAL JOURNAL *are listed in this department for the information of readers, and as acknowledgment and appreciation of their receipt from the publishers. Those of greatest interest will be selected for review in a subsequent number of the* JOURNAL.

THE PHYSICIAN AND THE LAW, by *Rowland H. Long*, with a foreword by *Milton Helpern*, M.D. 2d edition. 302 pages. Appleton-Century-Crofts, Inc., New York, N.Y., 1959. Price $5.95.

HEALTH MANPOWER SOURCE BOOK: Physicians, dentists and professional nurses, by Division of Public Health Methods in cooperation with Division of Dental Resources and Division of Nursing Resources. U.S. Department of Health, Education, and Welfare, Public Health Service. United States Government Printing Office, Washington, D.C., 1959. Price $0.50.

OFFICE ORTHOPEDICS, by *Lewis Cozen*, M.D., F.A.C.S. 3d edition. 430 pages; illustrated. Lea & Febiger, Philadelphia, Pa., 1959. Price $9.50.

SYSTEMIC LUPUS ERYTHEMATOSUS, A Mount Sinai Hospital Monograph, edited by *George Baehr*, M.D., and *Paul Klemperer*, M.D., with eight contributors. 84 pages; illustrated. Grune & Stratton, Inc., New York, N.Y., 1959. Price $3.75.

CLINICAL ORTHOPAEDICS, edited by *Anthony F. DePalma*, with the assistance of the associate editors, the board of advisory editors and the board of corresponding editors. No. 13, Spring 1959. 393 pages; illustrated. J. B. Lippincott Co., Philadelphia, Pa., 1959. Price $7.50.

FUNDAMENTALS OF NURSING, The Humanities and The Sciences in Nursing, by *Elinor V. Fuerst*, R.N., M.A., and *Lu Verne Wolff*, R.A., M.A. 2d edition. 662 pages; illustrated. J. B. Lippincott Co., Philadelphia, Pa., 1959. Price $5.

SCIENCE PRINCIPLES APPLIED TO NURSING, A Reference for Nurse Educators, by *Madelyn Titus Nordmark*, R.N., B.S., M.S. (N.E.), *Anne W. Rohweder*, R.N., B.S., M.N. Foreword by *Mary S. Tschudin*. 276 pages. J. B. Lippincott Co., Philadelphia, Pa., 1959. Price $5.

MOLECULES AND MENTAL HEALTH, edited by *Frederic A. Gibbs*, M.D. 189 pages; illustrated. J. B. Lippincott Co., Philadelphia, Pa., 1959. Price $4.75.

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